VOL. XI OLD SERIES VOL. LXXXVIII NO. 4

THE AMERICAN JOURNAL OF PSYCHIATRY

(FORMERLY THE AMERICAN JOURNAL OF INSANITY)

UNDER THE AUSPICES OF
THE AMERICAN PSYCHIATRIC ASSOCIATION

HOMEWOOD, BALTIMORE
THE JOHNS HOPKINS PRESS
JANUARY, 1932

Published Bi-Monthly — Subscription, $6.00 a Volume

Entered as second-class matter July 31, 1911, at the postoffice at Baltimore, Maryland, under the Act of March 3, 1879.
Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917. Authorized on July 3, 1918.

VOL. XI JANUARY, 1932. NO. 4

AMERICAN JOURNAL OF PSYCHIATRY

CLINICAL CONVULSIONS.*

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Too often our profession mistakes recognition of disease for its comprehension. To be able to classify certain happenings as an instance of manic-depressive psychosis or schizophrenia is not the same as appreciating the causative factors or even those integers creating the superficial appearances. To describe these is not to know them; and it is probable that these conditions no more than epilepsy are true diseases in themselves but are all of the nature of neural responses to noxious stimulation.

Indeed the instability of nerve mechanism in mental disease may be surmised from the frequency with which the history of psychoses is punctuated with paroxysmal disorders.

Hughlings Jackson has left us the notion of epilepsy as an occasional sudden excessive discharge of a nerve center, sensory or motor—to which we should add that such discharges are involuntary and are accompanied by some alteration in the stream of consciousness.

These emissions of energy are no primary disease unit but are merely the expression of an innate or induced instability of nerve centers of diverse origin operating probably through a common constant connecting mechanism—probably the cardiovascular system and its control. To seek for diverse origins imposes variety of direction in the search—allergy, circulatory disorder and chronic intoxication must all be brought under review. As there are many

* Presented at the eighty-seventh annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, June 1, 1931, Toronto, Canada.

causes there are many differences in the bearing and tendency of the condition; we must perceive the transient character of many of these episodes and rid ourselves of the idea that they are always followed at last by intellectual and emotional degradation.

Such a conception regards only a fragment of the spectrum of this disorder.

To understand the events in an epileptic fit, the nervous mechanism must first be regarded, then we should examine the evidence for disorders of blood supply accompanying the detonation, and lastly consider the nature of the factors beginning the sequence. We have already spoken of epilepsy as an involuntary discharge of energy from a nerve center accompanied by alteration of consciousness. The phenomena of the great fit can best be understood physiologically as a sudden cutting out of the highest level, the cortex, allowing the lower neuronic levels to pour downward an ungoverned stream of tonic postural impulses, in fact an abrupt decerebrate rigidity.

The following convulsions probably are the result of the gradual return of cortical influence, incoordinated and lacking volitional action.

The various initiating causes for these occurrences must operate as has been suggested through a common channel to produce so constant a pattern. I claim an identity of type and an inequality of degree in the different epilepsies, from the major seizures through petit mal to amnesic fugues to recurrent fainting and emotional storms originating from within and independent of environment. Among these should be included the voluminous mental states of unreality and those terrifying experiences called vaso-vagal attacks in which the patient is fear stricken, has a feeling of imminent death, a sense of enlargement of the limbs and losing definite contact with his environment, has an acute attack of cardiac hurry. This type of seizure may be a form of sympathetic epilepsy—all convulsions must be associated with an energy discharge over the sympathetic and autonomic systems and indeed include many more components than are indicated by externalized events. Probably the physiology of the great fit can be best understood by examining the minor attacks which are fragments of the fully developed seizure—just as we have fragments of the decerebrate posture produced by imperfect midbrain block. So we may see fragments of

the major attack when the vascular change has not been sufficient to cut out the entire cortex. Such a fragment was seen at Bellevue the other day: a young man for some years had suffered from a sudden giving way of his legs on laughing. He had assumed a constantly lugubrious countenance as a defence against this mishap. However, one morning during rounds he was sitting on the edge of the bed with his feet down and was made to laugh out loud by a remark of a negro nearby. Immediately, his hands were thrust out in tonic spasm, the face was tonically convulsed and cyanosed and he slid off the bed to the floor. During his fall the pupils were enlarged and fixed. He at once recovered and said he had not been unconscious. However, the fit was so brief that we could not disprove this statement though I most strongly doubt its correctness; nor could his reflexes be examined. Kinnier Wilson has found transient loses of the deep reflexes in such cataplexis. This form of convulsion is probably mesencephalic, the irritable center being in the thalamus. In all likelihood, the narcolepsies are subthalamic expressions of the same order. A sudden overpowering disposition to sleep descends on such patients, one of whom told me how he would be overcome in a directors' meeting in the middle of his own speeches, and more oddly still when kissing a girl in a taxicab.

It would be wearisome to detail all the types of epilepsy with psychic coloring and often proven focal pathology. One man in our wards recovered from explosions of furious choler with disorientation after the removal of bone compressing the left frontal cortex. Another improved but was not entirely freed of attacks of amnesic fugue after the elevation of an old fracture compressing the left temporo-occipital area. The mother of one of our medical students complained of attacks in which she was "dizzy and felt queer," at which times the left hand and forearm felt and appeared to her vision as being enormously increased in bulk: a right occipitotemporal meningioma was responsible for her attacks which were ended by its removal. Of course the most interesting and perhaps important psychic fits are those produced by organic disease of one or other temporosphenoidal lobes. These consist of the sensation of having lived through the exact scene then being enacted, the sentiment *du déja vu;* so acute is the sense of familiarity that a feeling of prescience exists, so that the subject feels as though he can foretell the next action and the next word that will be spoken.

To this voluminous mental state may be added "a peculiar thought" or a spectral word—heard projected in a world of unreal consciousness. Ghostly visitors appear in such states—phantoms appearing real but always known to be phantoms: one of my tumor patients would often see men and women in XVIIIth century court dress strolling across her New York apartment; a woman in Bellevue with left hemiplegia occasioned by a deeply placed right temporosphenoidal tumor would be terrified by the sight of a beckoning woman, dressed in blue, standing on her left hand. A complex visitant of another of my patients was a "little old woman who was dressed in stinking rags who kept ringing a bell,"—a projected thought associated with the crude subjective sensation of smell due to uncinate irritation. It may be noticed here that the results of uncinate irritation are always disgustingly unpleasant—probably a tribute to the severity of the disturbance—the pleasantest odor enormously multiplied would be unendurable.

It is an odd circumstance that such complicated projections as these are occasioned only by temporosphenoidal disease. One of my earliest patients, the wife of a Sussex farmer, in her attacks always saw the room filled with negroes—yet she told me she had never seen a negro in her life. However, she was the daughter of a sergeant stationed in Jamaica and was brought home to England at the age of two. Now each individual in his ascent from embryonic to adult life passes through the stages by which the race has passed. The lower animals make their chief intellectual contact with their environment through their hippocampal lobes, which in man are relatively small and unimportant. It may well be that these areas subserve early intellectual function which in later life is carried on by a higher mechanism. If this theory be true, then we may regard these apparitions as being involuntary resuscitations of early—perhaps infantile—memory patterns.

Not a few here must have experienced what Weir Mitchell sonorously called hypnagogic hallucinations—panoramic projections often of vision when just on the threshold of sleep. Persons so affected have each their peculiar variety. My own species is a spectral word—always a cliché—like "of course not," "yes surely"—heard dimly and without reality like a stage prompt. Then a second or two of normality comes—long enough for me to recognize the word as subconscious and tell myself to stand; then comes the silent

spring-shutter change in the mind—or the head—which breaks the thread of my previous thought so effectively that should I not sleep immediately I cannot recover the thread for several—five or ten minutes. This period of inactivity following involuntary energy release is a diagnostic feature of motor epilepsy, and helps properly to classify these events of the pre-dormitium as paroxysmal disorders, focalized probably in the temporal lobe. Myoclonic twitchings preceding sleep are probably of similar type. These considerations would show that thoughts—and their wayward and fantastic sisters, dreams—have dynamic neural patterns written by experience and released perhaps by altered physico-chemical conditions. Neural energy may travel by aberrant paths, the shunt being effected by trauma or disease causing resistance to flow of impulses.

Indeed, an extraordinary acuteness of reaction to sensory stimulation may occasionally be seen in epileptic persons.

Convulsions could be produced without fail in two patients under my observation: in one, a child, by flicking or sharply tapping the left side of the nose; in the other, an adult, by rubbing the dorsum of the left foot. The attack in the first case consisted in immediate dilatation of the pupils, tonic spasm of the arms and chest, respiratory arrest, cyanosis and slight frothing at the mouth, the whole lasting sometimes 15, sometimes 20 seconds, and ending in flaccid exhaustion after return to consciousness. There were no clonic movements in this child whom Dr. Jackson was good enough to come to the hospital to see and whose fit he regarded as a typical bulbopontine explosion.

In my second case, who was under the care of Sir Farquhar Buzzard, the convulsions were general and followed the usual procedure of a major seizure. The right parietal cortex was exposed in this case without anything of note being revealed.

Jackson reported a case of a boy whose seizure was precipitated by touching his head; a sharp tap or sometimes combing his hair would cause him to fall suddenly with paled cheeks but without loss of consciousness.

Some years ago, there was admitted to the neurological department of Bellevue Hospital, a boy aged 17 years whose history stated that for 12 months he had been subject to instant falling to the ground on hearing a sudden sound—a horse pawing the ground in a quiet street, a boy whistling behind him, would be enough to

produce a cramp in the left leg and a quick fall in which he was always bruised. He denied any loss of consciousness, and certainly to onlookers gave, for a long period, no evidence of such. However, after two years of these events, frequently repeated, he had a major convulsion which came on after the slamming of a door and was preceded, as were the other attacks, by a feeling of a cramp in the leg. These larger seizures did not entirely replace the fallings after sudden sounds. I induced both types of attack with ease, but did so rather seldom as he would fall violently on his face and, so innocent was he of any reflex of defence, I feared for his safety.

This is an example of the reflex acusticomotor epilepsy described by Oppenheim. For many months the boy was thought by some competent men to be hysterical. His first fall occurred at school when he was trying to solve a tough problem at the blackboard; his crayon broke and he fell sharply to the ground. His infantile history, however, supplied an organic background if not an organic explanation. Three weeks after birth there was still present a very large caput succedaneum over the right occipitoparietal area. This was opened and a blood clot evacuated. There was said to have been a bone defect in this area at that time. Symptoms of meningitis are said to have been present during the first two weeks of life. When five months old he had a general convulsion; later, he began to have jerky movements when falling asleep and on waking; in his mother's words, "the left leg would come up and the left shoulder and body go forward; the movement would occur eight or ten times but varied in number and intensity." These hypnagogic events lasted with lessening severity for 14 years; two or three years later they were replaced by the recurrent episodes just described.

The parietal cortex was exposed. A great thickening of the dura was discovered, produced by an old organized, subdural clot.

The attacks have become rare since the operation, and hypersensitiveness to sounds uncommon. This case has been told at some length because of its intrinsic oddness, but also because it shows how cerebral impulses can be short circuited by trauma and react quickly to stimulation by discharge to vasomotor changes.

Trauma, certainly, can render a brain area unstable. Severe skull and brain injuries in war were found to have given rise to subsequent convulsions in 4 per cent of 25,000 cases under review.

S. Bernard Wortis has shown in our laboratory that the injured cortex explodes with a smaller dose of a standardized convulsant (camphor) than does the intact cortex. Wilder Penfield (Brain, Vol. 53, part 2, p. 118) has pointed out the effect of scar tissue in cortical wounds, and believes that cicatricial traction on surrounding blood vessels is inevitable, and is the cause, by vasomotor reflexes, of the convulsion—the type of which will depend on the location of the scar.

Migraine has been reported in three patients as alternating with family periodic paralysis—but it would be futile to list here the numberless forms of equivalents which have been described, and are entirely familiar to this audience. Earlier in this address I used the term the spectrum of epilepsy. My general purpose has been to suggest that in this spectrum are many colors which merge insensibly into each other; uremia and eclampsia of pregnancy, the fits of general paralysis and brain tumor are there; the spasmophilic conditions of infancy are also to be thus grouped, including certain forms of tetany associated with alteration of consciousness; the chorea of pregnancy may be in this spectrum also, and—not very deep in the ultraviolet sector—are probably some cases of major hysteria, the motor phenomena of which suggest the temporary abandonment of a cortical for an infracortical mechanism.

In fine, as we have come to allocate destructive lesions of the brain by their focal signs, so we may associate the many types of epileptic expression with appropriate neural areas, which have been the objective of vascular attack; for example, petit mal is almost certainly an alteration of consciousness from frontal disorder, complex visual, auditory and psychical hallucinations from disorder in the temporosphenoidal lobes, gross color fits or twinkles from disorder in the occipital poles, myoclonic epilepsies from striatal implication, the cataplexis and narcolepsies from thalamic and subthalamic disorder, and the great fit as has been pointed out is a sudden momentary decerebration with a cutting out of the cortical totality followed by reviving paroxysm of the motor areas.

The researches of Bronson Crothers and Howard Smith (American Journal of Obst. Gyn., p. 19; 374, 1930) into the frequency of injury of the central nervous system, occurring in the act of birth illuminate an organic morbidity which has received insufficient attention. You will remember that in 100 spontaneous

deliveries there was bloody fluid in 11 per cent; in 50 low forceps deliveries 24 per cent of the cistern fluids were bloody. In five cases of breech extraction there was blood in 40 per cent of the fluids, and when in five cases the first stage of labor had lasted over 24 hours it was found that 60 per cent of the fluids were contaminated with blood. The fluids of Cæsarean babies were all blood free. Such birth injuries must often be capable of lowering the threshold of neural explosion to a point accessible to stimulation by metabolic products, toxic maybe, but innocuous in a subject with a higher threshold of nerve cell irritability.

The act of birth assumes then a highly hazardous and romantic aspect. Death has been called the greatest adventure of life, but there is no adventure in a certainty; the real gamble for all of us lies in our entrances, our future lies behind us, and with this knowledge we may be able to do good preventative medicine even in the field of epilepsy with assistance from our obstetrical colleagues.

Once the minute injury has been done our efforts in treatment must reasonably be directed, first to raising the threshold of neural explosion and second, to getting rid of all detonating stimuli likely to reach it.

The first is achieved by the continued use of small doses of neural sedatives, bromides of course, but still better, phenobarbital. Pavlow (Conditioned Reflexes, 1927, 10) has taught us how inevitably the neural reflexes become conditioned and to allow fits to occur because of fear of "drugs" is obsessional superstition.

The next is to rid the oversensitive organism of noxious stimulation. To this end have been directed most of the modern therapeutic systems. Lane and Reed, when permitted, enthusiastically resected the entire colon; other men more conservatively have been content with a bacteriological revision of the great intestine, by providing proper diet, peristalsis and flora. Geyelin in 1921 studied the effects of periodic starvation on epileptics and found that severe fasting considerably reduced the number of attacks and in a small number eventually caused their arrest. From his work has been evolved the plan of producing by a diet very rich in fats, a ketosis similar to that produced by starvation. A child of 58 pounds on this system should begin by 60 grms. of carbohydrate, 129 grms. of fat and 25 grms. of protein in the 24 hours.

No one program, however, will be successful or even moderately successful in all cases. Nor could we expect one design to be of service against so varied a congery of symptoms.

An approach to real knowledge may lie in the chemical study of sympathetic nervous system stimulants and toxic protean split products produced by proteolytic ferments.

The humoral pathology—to use Zabriskie's phrase—of the disease must be made comprehensible by anaphylactic studies; and when these things have all been added unto us we must remember to treat the patient as a whole, to adjust his environment and his problems, to arouse his interests, keep fired his zest for living, inculcate discipline and wise habits, and as far as possible allow him to live normally within himself—with fair weather in his soul—and as happily as maybe also in the society of his choice.

EXPERIMENTAL CONVULSIONS.*

By S. BERNARD WORTIS, M. D., New York.

In the Bellevue Hospital Laboratory of Experimental Neurology, we have been interested in some of the problems associated with convulsive disorders. I might say at the outset that we regard epileptic convulsions as having a physico-chemical, *i. e.*, organic basis. The more important approaches to the experimental study of fits have been along the following lines:

(1) Changes in brain vascularity:
  (a) Cerebral anemia.
  (b) Cerebral engorgement.
(2) Traumatic stimulation of the central nervous system.
(3) Chemical stimulation of the central nervous system.
(4) Electrical stimulation of the central nervous system.
(5) Changes in intracranial pressure.
(6) And combinations of all these.

A brief review of some of the more important findings will be timely here:

(1) The earliest work on cerebral anemia was done by Kellie (1824) and Piorry (1826), who were able to produce convulsions in animals by bleeding them. Later Astley Cooper (1836), Kussmaul and Tenner (1857), Leonard Hill (1896), Stewart and Pike (1909) and Gildea and Cobb (1930) were able to produce convulsions in animals which were subjected to cerebral anemia. Cobb reported that "unlike some investigators convulsions in our cats usually occurred during the period of occlusion as well as later, after the blood was allowed to return to the cortex; this may be explained by the fact that light ether anesthesia was given and the animals were allowed almost to regain consciousness before the arteries were ligated."

* Presented at the eighty-seventh annual meeting of The American Psychiatric Association, Section on Convulsive Disorders; Toronto, Canada, June 1, 1931.

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Landois (1867) and Herman and Escher (1870) were able to produce convulsions in animals by compression of the neck veins with resulting partial asphyxia. Pike (1931) reported an increased susceptibility of animals to convulsions following ligation of the external and internal jugulars and also after blocking of the cerebral transverse sinuses. His animals moreover did not develop spontaneous convulsions.

(2) The rôle of head trauma in the production of convulsive phenomena is well known. Brown-Sequard (1851) traumatized various parts of the central nervous system of rabbits and induced convulsions. Nothnagel (1868) and Westphal (1871) were able to obtain convulsions in rabbits after brain puncture or gross head injury. Subsequently Luciani (1878), Vulpian (1885), Goltz (1892) observed convulsions in cats and dogs following various cortical injuries. Barbour and Abel (1910), Thomas (1921) and Syz (1923) have demonstrated in the frog that the injured central nervous system stained deep pink after injections of acid fuchsin (in sufficient quantity to cause convulsions) while in the intact animal the brain remained uncolored with similar dye concentration. Sauerback (1913), Dandy and Elman (1925), Wortis and McCulloch (1931) have been able to demonstrate a heightened sensitiveness of the animals to a standard convulsant following cerebral laceration and skull fracture. The histopathological results of brain laceration in the production of cerebral scarring has been demonstrated by Del Rio-Hortega (1927), Foerster (1930), Penfield (1927, 1930), Stevenson and Wortis (1931). A surgical method for the relief of certain types of post traumatic epilepsy has been devised by Foerster and Penfield.

(3) The production of experimental fits by use of various chemical substances dates back to 1864, to Morcé who first used absinthe for this purpose. Later Magnam (1876), Victor Horsley (1885), Hughlings Jackson (1885), Boyce, Russell, Purkinje, Muskens and others have used various drugs and attempted to explain epileptiform phenomena on the basis of their results.

Focal chemical stimulation of the cerebral cortex has been done with many different substances, placed directly on the exposed brain. Landois (1887) used kreatin and other urinary products; Bickel (1898) used bile pigment; Korangi and Tauszk (1890) used Leibig's meat extract; Baglioni and Amantea (1914) used strychnine. All the aforementioned workers were able to obtain focal

convulsions related to the placement of these substances, noxious to the brain tissue. Amantea (1921) further demonstrated that following cauterization of the cortex identical stimulation did not induce convulsions. Recently at the Bellevue Laboratory we have succeeded in standardizing a solution of camphor monobromide and used this for the production of experimental fits. Some of the commonly known substances capable of producing typical clonic and tonic convulsions in animals are: absinthe, camphor, certain convulsant dyes (described by Macht, 1912), cocaine, strychnine and caffeine in large doses. Several of these drugs we have standardized in minimal convulsive units required per pound (or per kilogram) of the animal weight.

(4) Electrical galvanic cortical stimulation was done as early as 1870 by Fritsch and Hitzig. Later Ferrier (1873) at Hughlings Jackson's direction was able to produce focal convulsions by this method. Victor Horsley later used the same method to chart excitable brain areas, Munk (1881) and recently Sparks (1927) showed that convulsions could be elicited by cortical electrical stimulation. Sparks also showed that electrical stimulation with weaker currents produces clonic, and with stronger, tonic movements. Also that the electrical irritability of the brain was increased by ketone, thujone (a camphor isomer) and low blood sugar level, and decreased by phenolbarbital. Very recent work by Lepicque (1926) and Bourguignon (1923) on the study of tissue chronaxie is affording us most valuable information. They have shown that as a rule the more excitable tissue has a shorter chronaxie, a shorter period of action, a shorter refractory period and quicker rate of propagation than a less excitable one.

(5) The effects of increased intracranial pressure in causing greater susceptibility to convulsive seizures have been excellently reviewed by Temple Fay. Work by Fay, Winkelman, Weed, Rowntree, Kubie, Elsberg and Pike has shown the apparent rôle of "hydration states" in the production of epileptic seizures. Lennox and Cobb (1928) have emphasized the following conditions as predisposing to a convulsive seizure:

(a) Poor oxygen supply.
(b) Increased permeability of tissues.
(c) Edema.
(d) Alkalosis.
(e) Increased intracranial pressure.

Foster Kennedy and Wortis (1931) have recently reviewed the methods for the relief of increased intracranial pressure in the more common pathological conditions.

I. The Standardized Convulsant.

At the Bellevue Hospital Laboratory for Experimental Neurology we have used camphor monobromide to produce experimental convulsions in cats. The exact mechanism of the convulsive action of this drug is not known, but evidence is accumulating that its action is due to nerve cell irritation. The standard solution is made by dissolving 10 grams of camphor monobromide (U. S. P.) in 100 c. c. of 95 per cent ethyl alcohol. This solution is then injected into the femoral vein, starting with a definite amount per pound of animal and uniformly increasing this amount at equally spaced time intervals, until the animal has a generalized convulsive seizure. This was regarded as the minimal convulsive dose. Normal control animals require .018 c. c. to .026 c. c. of this solution per pound to produce a generalized fit.

The similar characteristics of a true epileptic fit occurring in human beings and in animals (cats) under suitable dosage of camphor monobromide are as follows:

(1) There are rapid disordered convulsive discharges occurring in different parts of the body (especially seen in myoclonic types of epilepsy).

(2) Transient episodes of petit mal—associated with periods of temporary confusion, occasionally associated with eneuresis and some pupillary dilatation. These small attacks are difficult to determine in animals; but a cat can often be observed to stare about, have a fleeting pupillary dilatation and coincident eneuresis.

(3) The great fit—associated with loss of consciousness, pupillary dilatation, motor convulsive seizures and loss of sphincter control; all of which are seen in the cat under adequate dosage of camphor monobromide.

(4) Loss of reflex excitability in the period directly following the great fit. The superficial and deep reflexes are not elicitable in this stage.

(5) Frothing at the mouth; sweating of the body (foot pads in animals); respiratory alterations; and occasionally biting of the tongue are observed.

(6) Persons suffering with epileptic seizures, and animals given camphor monobromide in oil by stomach, have a tendency to develop fits at the periods of awakening or falling asleep.

From the foregoing one must deduce the discharge of stimuli during a convulsive seizure as an energy outflow through all neural mechanisms.

II. The Effects of Various Brain Lesions on Experimentally Induced Convulsions.

A. FRONTAL CORTEX ABLATION.

It has long been known that extensor rigidity often followed cerebral cortex ablation. Sherrington (1896), later Thiele (1905), and Weed (1914) investigated the genesis of this resultant hypertonus. Weed (1914) showed, and Cobb, Bailey, and Holtz (1917) confirmed his findings, that stimulation of the mesial portion of the cerebral crura inhibited the rigidity brought on by decerebration. Bazett and Penfield (1922) aseptically decerebrated cats and found that tonic extensor rigidity occurred in the contralateral, and tonic flexor rigidity in the ipsolateral, limbs. Subsequent work by Warner and Olmstead (1923) showed that if only the electrically responsive motor area (cortical) of a cat was cut away no increase in extensor tonus ensued; but if the cortex, anterior to this region was removed, definite hypertonus in the contralateral forelimb and hind limbs resulted. This increased tonus was produced even in the presence of an intact motor cortex.

By the method described by Langworthy (Bulletin Johns Hopkins Hospital, Vol. 42, p. 32) the area frontalis of the cerebral cortex of the cat was removed bilaterally. The abnormal, enduring extensor hypertonus of the legs was noted and persisted during the animal's life (at least four months). The tonic postural alterations noted by Langworthy were confirmed. This animal when given camphor monobromide by vein 2 hours, and 2, 7, and 74 days after operation, had a generalized clonic convulsive seizure.

B. MOTOR CORTEX ABLATION.

The left motor cortex (*i. e.*, anterior and posterior sigmoid gyri) outlined by Langworthy and Weed was excised with aseptic technique in several animals. They were allowed to recover and at varying periods after operation were given convulsions by the intravenous injection of camphor monobromide. There was motor disability in the contralateral limbs but the cat was able to walk quite well. The convulsions elicitable within periods of 7 to 10 days after such ablations are clonic in the limbs on the same side with the lesion and tonic in the contralateral limbs. After 10 days

clonic phenomena in the contralateral limbs again appeared. These data correspond with work by Pike (using absinthe) and it seems reasonable to infer that parts of the nervous system (the extra-pyramidal system), in the midbrain or below this level are sufficient for the genesis of tonic convulsive phenomena.

C. PARIETO-OCCIPITAL LOBE ABLATIONS.

In several animals the left parieto-occipital cortical substance was removed anatomically, with aseptic technique, without injury to the underlying thalamic region. This included gross removal of the ectosylvian, suprasylvian, posteriorsylvian, marginal and posterior composite gyri. The animals thus operated upon showed several characteristic alterations and have been described in a previous publication.

All these cats have generalized clonic convulsive seizures on the intravenous administration of the standardized convulsant at any time following the operative procedure, providing the anesthetic effects have worn off.

D. CEREBELLAR CORTEX.

In another group of animals ablations of the left cerebellar lobe resulted in the characteristic signs attributable to unilateral cerebellar destruction. All these cats have generalized clonic convulsive seizures on the administration of camphor monobromide by vein, directly after operation, or at remote post-operative periods.

E. STRIATAL LESIONS.

In a series of cats in which left striatal lesions were experimentally produced by Wortis, Pike and Coombs it was demonstrated that destructive striatal lesions do not prevent the occurrence of typical convulsive phenomena (produced by standardized convulsants, camphor monobromide or absinthe) providing the motor cortex and the cortico-spinal pathways are intact.

F. DECEREBRATION.

Several animals were decerebrated by the method described by Hinsey, Ranson and McNattin (Arch. Neurol. and Psychol., Vol.

23, p. 1, January, 1930), *i. e.*, in planes passing from the rostral border of the superior colliculi,

(a) Through the optic chiasm.
(b) Just caudal to the mammillary bodies.

The behavior of these animals after operation was found to correspond with those with similar lesions described by the aforementioned investigators. Four hours after section from the superior colliculi through the optic chiasm the animal was able to walk about the laboratory.

Camphor monobromide given to these cats directly following injury, or within 24 hours, fails to elicit any clonic convulsive phenomena although tonic extension of all limbs obtains and is associated with marked stimulation of the respiration.

This finding is particularly interesting as regards the place of origin of clonic convulsions. It has been stressed elsewhere that in these cats some of the prespinal centers which remain are the red nucleus, the hypothalamic centers, the substantia reticularis, the tectum, the cerebellum and the vestibular system. Evidently these are sufficient to carry impulses through the complex coordinating mechanisms necessary for complete reflex walking but are not able to produce clonic convulsive phenomena by the injection of a convulsant drug within 24 hours.

These findings confirm the observations on motor cortex ablation which have already been described.

III. The Effects of Vascular Lesions.

The effects of these lesions on the animals' subsequent susceptibility to convulsions have in part been described. It has been shown that sudden cerebral anemia and also its sudden amelioration give rise to clonic and tonic epileptic seizures.

The related work of Pike, Elsberg, McCulloch and Rizzolo must here be noted. These investigators found that prolonged cerebral anemia resulting in cortical cell destruction yielded results similar to motor cortex ablation; whereas temporary anemia resulted in an increased susceptibility to absinthe. These data point to the requirement of living excitable motor cortical cells for the production of clonic convulsive phenomena after injury.

IV. Cervical Sympathectomy.

Cervical sympathectomy has in recent years occasionally been heralded as a suitable operation to relieve epilepsy, on the assumption that with the removal of the cervical sympathetic chain the cerebral and meningeal blood flow to the brain would be increased and thereby relieve vasospasm and cerebral anemia with the resultant fit. Kussmaul and Tenner (1859) were able to cause convulsions in a rabbit in which one carotid was tied, by faradization of the cervical sympathetic on the opposite side. Meagher and Ingraham (1927) were unable to confirm this in rabbits and cats. Alexander (1889) and Wagner (1925) did cervical sympathectomy on epileptic patients with disappointing results.

In a few animals we have resected small portions of the cervical sympathetic trunks on one or both sides and subsequently given the cats camphor by vein. These cats can be given generalized clonic convulsions, with doses of the solution that are similar to those required for intact control cats. This series as yet is too small to be definite as to the amount of convulsant drug required to elicit fits; but it is important to report that convulsions can still be elicited despite cervical sympathectomy. Also that in the animals having but one cervical sympathetic chain removed, the convulsions when induced start simultaneously on both sides of the body!

V. The Effects of Adrenalectomy.

The effects of adrenalectomy on subsequently induced absinthe convulsions have been described by Coombs, Pike and Wortis. It was found that the minimal convulsive dose was lowered from 33 to 50 per cent by adrenalectomy and the lethal dose was more strikingly reduced. These findings are interesting in relating the rôle of the adrenal gland and the sympathetic nervous system, in epileptic convulsions.

VI. The Effects of Extradural Bodies Pressing on the Brain.

In a series of cats extradural sterile bodies (sterile waxed cotton pledgetts) were placed inside the skull to compress various parts of the brain. The effects of such lesions on subsequent convulsions

induced by camphor varies somewhat with the location of the foreign body and the time elapsed between the operation and the induced convulsion. Suffice it to say here that such lesions, varying from 2-4 per cent of the intracranial volume, introduced over parts of the brain other than the motor cortex do not prevent the occurrence of clonic convulsive phenomena on the intravenous injection of camphor monobromide. Foreign bodies pressing directly on the motor cortex of one side yield results that resemble those following surgical removal of the corresponding area. That is to say, the induced convulsion is more tonic on the side opposite the lesion, and is clonic on the same side with the lesion. Also, the animal during the seizure has the tendency to turn itself from the side of the lesion. This is likewise observed in convulsions following corresponding motor area ablation experiments. These findings confirm the work of Pike and Elsberg, who also showed that after six weeks to two years the brain underlying such foreign bodies may show an increased sensitiveness to a convulsant drug (absinthe).

VII. The Effects of Trauma.

Head trauma has been known to produce convulsions. In a series of 24 animals subjected to head injury Wortis and McCulloch (1931) have demonstrated an increased sensitiveness to camphor coming on four days after trauma and persisting over four months or more. This may be caused by blood in the subarachnoid spaces or brain laceration.

VIII. Discussion.

These experimental data bear evaluation with a view to the problems of (1) localization of tonic and clonic convulsive components; and (2) significance in relation to studies in clinical epilepsy.

Victor Horsley (1885) showed that convulsions (following absinthe administration to animals) were either clonic or tonic; the former depended on the functional activity of the cerebral motor cortex, and the latter on its absence. Ziehen (1886) also showed that motor cortex ablation resulted in a temporary loss of the clonic motor events; only tonic spasms were obtainable. Hill (1900) by occluding the cerebral circulation after absinthe injection was able to instantly transform convulsions from the clonic

to the tonic form. However it is well established that clonic convulsions may arise from the lower (and phylogenetically older) mechanisms after injury to higher cerebral units. This moreover does not argue that in the intact animal such lower centers are the origin of clonic convulsions. It seems fair to deduce that in the intact animal the cortico-spinal mechanism is the most important (and probably the neural mechanism most easily stimulated by camphor and other toxic substances)—and probably gives rise to clonic convulsions. When this neural hierarchy is injured lower motor mechanisms may take over its function after a short period of energy flow readjustment. Hughlings Jackson's conception that there is a change in the quantity of nervous energy passing through the remaining neural pathways or levels of the nervous system after an injury to one of them accounts, in great measure, for some of the remarkable recoveries (always more or less incomplete). This has been emphasized by Pike who pointed out that with this hypothesis one need not employ the overworked hypotheses of "inhibition" and "shock" (of Goltz) to explain these phenomena.

One is tempted to relate some of the more important factors already noted, to clinical conditions. It becomes clear that probably the reason one does not at times see focal convulsions associated with large endotheliomata pressing directly on the motor cortex is because the functional activity of this region is impaired by the tumor. Also adrenalectomy does not strike one as a very safe or a very rational procedure in cases of epilepsy. Cervical sympathectomy can never be an adequate cure for epilepsy so long as the toxic neural detonating substances (which probably cause some cases) remain active in the individual. Severe head trauma undoubtedly is a contributing factor to the facilitation of convulsions.

I cannot close this presentation without emphasizing this idea—that during a convulsive seizure there is a discharge of stimuli (*i. e.*, energy) which flows out through all available neural mechanisms, *i. e.*, via the cerebrospinal and vegetative nervous pathways. The discharges over the cerebrospinal system resulting in the motor phenomena; those over the autonomic nervous system producing characteristic sympathetic "discharge reactions" which probably in great part constitute a factor of safety for the maintenance of homeostasis in the organism.

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DISCUSSION.

Dr. F. H. Pike (New York City).—Having done some of this work in conjunction with Dr. Wortis, I can confirm it. Having done some of it individually, I can also confirm it.

One thing that I would like to emphasize is that in these experimental animals we must eliminate pain. We want reactions that are due to the drug or other experimental measures employed and not reactions that are due to pain.

That was the reason for the use of novocain even for trifling operations, even for injection into the vein when nothing else is done. That is the reason why the abdominal viscera and peritoneum are so freely injected with novocain in all these experiments.

There are one or two things of which I would like to speak. I have read in the literature about convulsions following operations on the central nervous system. I have done a number of them. I don't know what is wrong with my technic, but I never have seen convulsions in animals following any cerebral operation.

In one cat in which there was a convulsion before operation, I never noticed any afterwards. The result has been a bit disappointing, in one sense, not disappointing in others.

In the matter of cervical sympathectomy, this operation has no effect on the ease with which convulsions are elicited, except that it perhaps takes a lower dose at certain seasons. I am beginning to think there may be a time element there. It may take a little higher dose in a certain period after removal of the cervical sympathetic on one or both sides, whereas apparently later on even less may be required.

From the experimental point of view at least, cervical sympathectomy has nothing to recommend it to me.

I feel the same way about adrenalectomy. Unilateral removal of the adrenal has practically no effect on cats. I have had some operated animals around the laboratory now for five or six months. Two of them are in the same cage together and indulge in a boxing match every day. They have gained in weight. They have kept in very good condition.

Unilateral removal of the adrenal would have little to recommend it. To take out both adrenals, I do not believe would do the patient much good.

Later on I shall have something more to say about the sympathetic aspects of this in the cardiovascular and other reactions.

Dr. Wortis.—I want to thank Dr. Pike for his kind expressions and encouragement. Only last week, at the meeting of the American Neurological Association, I presented some experimental work on brain trauma. In this group of animals which were subjected to laceration of the brain or to the introduction of blood in the subarachnoid space (both conditions might well obtain as a result of injury to the head)—I observed spontaneous convulsions in only one animal.

STUDIES IN THE BLOOD VOLUME OF EPILEPTICS.*

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We recognize epilepsy to be a symptom-complex associated with a variety of superficially unassociated conditions. One cannot but be impressed by the marked differences in the pathological findings in the brain at autopsy. Convulsions may be brought on experimentally by injury to various portions of the brain or cord. Indeed, there is hardly a location in the central nervous system in which trauma in some form, whether due to a blow, a chemical irritant, or electricity, may not bring about a convulsive state. It is interesting to note that injuries, in themselves not productive of convulsions, render the animal more susceptible, as has been shown by the fact that smaller amounts of absinthe produce convulsion in such experimental animals.[1]

Clinically, similar pathological conditions are not always accompanied by epilepsy. For example, it is not a constant feature in microcephalics or in hydrocephalics. Not all brain tumors cause convulsions although they are present in some cases. Following inflammatory conditions of the meninges, there may be epileptic attacks but they are by no means constant. Apparently similar traumatic or degenerative processes in the brain may or may not be associated with convulsions. We conclude that no specific lesion of the nervous system has been satisfactorily demonstrated in epileptics. Convulsions occur in only a relatively small number of persons with gross brain lesions.[2]

Aside from changes in the nervous system various conditions have been proposed as factors in the causation of convulsions. Gastro-intestinal disturbances,[3] dysfunction of the various endocrine

*Read at the eighty-seventh annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Toronto, Canada, June 1, 1931.

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glands, such as the thyroid,[4] and parathyroids,[5] the adrenals,[6] the pituitary gland,[7] and a combination of glandular disorders have been considered as causes of epilepsy.[8] Reed [9] described a specific bacillus in epilepsy but this has not been substantiated by others. A specific toxin has been described by Ceni.[13]

Aside from studies on the brain, spinal cord, and endocrine glands, a great deal of research has been done on the blood, urine, feces and spinal fluid. On close analysis, although there may be wide differences between individual patients, essentially normal values [2] were found in the estimations of the red cells, white cells, blood grouping, hematocrit readings, coagulation time, blood calcium, Wassermann reaction, blood nitrogen, both non-protein and urea nitrogen, blood sugar, and blood chlorides. There were higher values in the fibrin content of the blood.[10] Agreeing with this finding is noted increased sedimentation of the red cells.[11] The positive findings of increased fibrin and speed of sedimentation in a number of patients may be evidence of an unusual degree of tissue destruction in these patients.[2]

It is suggested from this rather casual review of proposed etiologic factors that the tendency to convulsive reactions on the part of the nerve cell may well be an inherent property beyond present analysis. We are confronted with the idea that individual variations in the manner of reacting to environment is characteristic of living material, and in the presence of a given stimulus only certain individuals may react with convulsions. The words of Kussmaul and Tenner [12] are as applicable now as in 1859 when they said, "Every physician of the present day who is at all judicious will relinquish the hope cherished with child-like confidence, by certain schools and times, that pathological anatomy is destined to give an explanation of the nature and seat of epilepsy, and he will only expect that result from the progress of experimental physiology of the nerves."

With apparently so indefinite a knowledge of the causative factors, it would seem that a discussion of treatment must be entirely theoretical and empirical. However, instances of successful empirical treatment of disease before the etiology is definitely understood are not uncommon. We may cite, as examples, quinine therapy in malaria, mercury and iodides in syphilis, chaulmoogra oil in leprosy and today's liver therapy in pernicious anemia.

Indeed, in some conditions, subsequent discovery of the specific cause but confirmed the treatment.

There have arisen in the last few years several methods of treatment of epilepsy which have been to a greater or less degree successful in decreasing or controlling convulsions. Among them may be named:

(a) Starvation treatment.[14]
(b) Ketogenic diet.[15, 29, 30]
(c) Dehydration by water intake limitation.[16]
(d) Ingestion of the acid forming salts, such as calcium or ammonium chloride and ammonium nitrate.[17]
(e) Intravenous glucose, magnesium sulphate or hypertonic salt solutions.[18]
(f) The surgical procedure of colonectomy.[19]
(g) Induction of fever.[20]

On careful analysis, these procedures seem to have one effect in common—dehydration. The question arises whether the beneficial effect derived from these various methods may not be dependent, at least to some extent, on this common factor. Let us analyze the methods enumerated: (a) Gamble[21] presents data which indicate that in fasting there is produced a change in the body fluid in the direction of reduction. He further states that there is thus presented the possibility that this change in fluid volume may be the event which is beneficial in epilepsy rather than the change in body fluid reaction (acidosis) which fasting also produces. Convulsions may occur in the presence of ketosis such as is present in diabetic coma (Lubbe, 1920) and in the cyclic vomiting of children; (b) In a study of the mechanism of the ketogenic diet Bridges and Iob[22] state that the hypothesis of ketosis and acidosis in explaining the beneficial effects of the ketogenic diet in epilepsy can no longer be held valid, and suggest that the improvement following this diet is associated with removal of the surplus sodium or extracellular fluid from the body. Benedict and Milner (1907) have shown that all other conditions being constant, a sudden change from a high carbohydrate to a high fat diet may be associated with a marked loss of water and salts from the body; (c) In dehydration by fluid limitation, one may logically conclude that there takes place a decrease of the body fluids; (d) Ammonium chloride and nitrate and calcium chloride have been used successfully as dehydrating agents in edema of cardiac and nephritic origin and in ascites. Lennox and Cobb[2] found a temporary beneficial effect of these salts

in the convulsive seizures of epileptics. They state that acidosis is produced by an increase in the blood chlorides. We shall show in our studies that an effect of administration of these salts is a lowering of the blood CO_2. Nevertheless, with acidosis present seizures may occur as previously. Lennox and Cobb have found on the 10th day of ingestion of these salts, with acidosis present, convulsion may return in a flood. Their explanation, that increased concentration of the chlorides in the tissues counterbalanced the beneficial change in the Ph, suggests to us that this concentration of chlorides in the tissues favors the retention of fluids. Our own results with ammonium nitrate therapy do not show an increase in the plasma chlorides; however, this does not preclude the accumulation of chlorides in the interstitial tissues. In the treatment of the edemas with these salts, success is dependent, to some extent, on the amount of fluid ingested and the fluid intake during such treatment is limited, ordinarily, to 1000 c.c. per day. Perhaps the conflicting reports[22] on the results of treatment of epileptics with the acid-forming salts may be dependent on the fluid intake; (e) Hypertonic solutions of glucose, magnesium sulphate or 25 per cent to 30 per cent saline solution, are used as dehydrating agents to relieve intracranial pressure. Since there is a definite chemical difference in the agents used, their similar effect of dehydration suggests that this is the common factor; (f) Colonectomy was looked upon at one time as a procedure of some merit in epilepsy. The feces in the small intestine are quite fluid. In the colon, a large portion of this fluid is absorbed. In many of the patients, reasonably free from convulsions after colonectomy had been performed, there were present frequent watery stools.[23] Again we have a probable factor of dehydration. A surgical procedure done under an anesthetic carries with it a temporary fasting state which we have already shown is accompanied by dehydration. In many instances it has been noted that temporary relief from convulsions occurred after appendectomy or other types of operation; (g) One of us (G) has shown that during acute illnesses accompanied by fever there is a reduction in the number of convulsions. Clinically, there may be present some degree of acidosis during fever but there is also a factor of dehydration. Edgeworth,[24] Miller,[25] and Spangler[26] reported favorable results from non-specific protein therapy. The mechanism for improvement may have been the fever. The added

effect of vasodilatation must be given consideration. This effect of protein shock has been demonstrated by Allen, Smithwick, and others by work in diseases of the extremities caused by vasoconstriction.

On summing up the enumerated therapeutic agents or methods used to modify the course of epilepsy, it does appear that dehydration is a condition common to all. We do not conclude that this is the only factor but we wish to emphasize it as a factor in common.

Lennox and Cobb,[27] in a discussion of the relation of certain physicochemical processes to epileptiform seizures, present three theses: (1) Acidosis tends to inhibit and alkalosis to augment seizures; (2) Increased tension of oxygen in the tissues tends to inhibit and decreased tension to augment seizures; and (3) edema of the brain tends to increase and dehydration to decrease seizures. Analyzing these three theses, one is impressed with the marked inter-relation of the conditions which decrease or increase seizures.

Anoxemia results in the passage of fluids outward through the capillary walls and makes for tissue edema; the reverse is true of acidosis. The latter favors the passage of fluid toward the capillaries and tends to produce tissue dehydration. Acidosis produces an increased oxygen tension. The chemistry of the blood is such that in the presence of acidosis oxygen is more readily given up by hemoglobin and more oxygen is available for us by the body tissues. The reverse is true in anoxemia. There is, then, an increased oxygen tension favoring tissue dehydration in acidosis, and a decreased oxygen tension favoring edema in anoxemia. The favorable factors as enumerated in the three proposed theses are practically interchangeable, as also are the unfavorable factors. In the final analysis, one gathers the impression that conditions favoring tissue dehydration limit seizures while conditions favoring edema increase seizures. Acidosis, increased oxygen tension, and dehydration are factors in common, as are alkalosis, decreased oxygen tension, and edema. These changes in the chemistry of the blood are closely associated with dramatic vasomotor phenomena. Acidosis favors vasodilatation and alkalosis favors vasoconstriction. Acidosis tends to inhibit the vasoconstrictor response to adrenalin,[31] while alkalosis increases the sensitivity of the vasoconstrictors to this agent. The regulation of intracranial circulation which is delicately and effectively balanced under normal conditions is demon-

strably effected by the chemical composition of the blood. The influence of physicochemical changes on vasomotor control, and other features of circulatory dynamics have not been studied adequately in relation to epilepsy and offers an interesting field for research.

McQuarrie,[22] in his summary, states that the superficial relationship between water balance and occurrence of seizures has been determined under various conditions in a number of epileptic children. There appears to be a tendency in epileptic subjects to retain water during the active phase of the disease. He further states that convulsions tend to occur when a positive water balance above a certain magnitude is established. Also, in experimental work, Rowntree has shown that large quantities of water administered to animals cause water intoxication and convulsions.

At this point we may well mention three diseases in which, as a result of a survey, we have found the incidence of epilepsy to be comparatively low. Talbot [29] states: "The co-existence of epilepsy and diabetes is so rare that it hardly seems possible that the two conditions can depend on any common etiological factor. Their character differs so fundamentally that it seems far from improbable that there might be features in one disorder which would be curative for the other and vice-versa. For example, in diabetes, there develops ketosis and other metabolic disturbances which appear to reduce the severity of epilepsy." He quotes Joslin that, "among 5091 true diabetics whom I have seen, there has been no case of epilepsy."

In reversing the inquiry as to the incidence of diabetes among epileptics, rather interesting data were found. At the Monson State Hospital, during the past 30 years covering observations on about 6325 cases, one of us (H) has noted but one case of diabetes. It is an interesting fact that this patient, after developing diabetes, showed a decrease in the frequency of convulsions and during the later months of his life, was practically free from seizures. This case occurred before the insulin era, when ketosis and dehydration were common in diabetics. In answer to a questionnaire covering a total of 13,597 epileptics, only seven cases of diabetes were reported, which is an incidence of 1 to 1944. Among 5442 non-epileptic feeble-minded the incidence of diabetes was 1 : 1088. To get the true significance of these numbers we should keep in

mind the finding from statistics gathered by Davenport [33] during the World War, that the incidence of epilepsy among 2,500,000 drafted men was 1 : 194. One of us (H), in a survey of Hampden County, Massachusetts, found an incidence of 1 : 400.

A low blood volume has been found in hypothyroidism.[34] For this reason we felt it advisable to gather statistics as to the incidence of epilepsy in cretinism. In a survey of 8622 institutionalized epileptics, seven cretins were reported, making an incidence of 1 : 1231. For comparison, a survey was made of 5442 non-epileptic feeble-minded subjects. Here we found 18 cretins, an incidence of 1 to 302, or slightly over four times the frequency in epileptics.

A low blood volume has been reported by Silbert [35] in thromboangiitis obliterans or Buerger's disease. In a survey of about 2600 cases with this disease, not one case of epilepsy was reported.

It is our impression, based on the above data, that epilepsy is less common in diseases which favor dehydration, or in which a low blood volume is present. We do not mean to infer, however, that dehydration and low blood volume are interchangeable terms.

In analyzing the seizure, we must recognize that a dramatic change of some kind takes place in the brain, precipitating the convulsive attack. Edema alone seems to produce seizures as is evidenced by the fact that seizures occur in animals which have been overloaded with fluid, in eclampsia, and in fracture of the skull, where edema plays an important part. Dehydration by hypertonic solutions has been used successfully by Fay, Kennedy and Wortis in the prevention of seizures in such cases.

We know that anoxemia alone produces convulsive seizures, as shown experimentally by Lennox and Cobb,[2] and dramatically in Stokes-Adams syndrome where, as a result of cardiac standstill, there is complete cessation of oxygen renewal. Here may be stated that in observing normal individuals who have had severe fainting spells, two of us (H and G) have noted that just before regaining consciousness there have occurred moderate clonic contractions. In this condition because of temporary slowing of the circulation, a condition of anoxemia of the brain exists. Oxygen consumption by the brain tissue when compared with other body tissues is quite high and the oxygen lack may explain seizures in such unrelated conditions as polycythemia, sudden anemia, and hypoglycemia. In

the presence of increased oxygen, strychnine convulsions are prevented. The reverse is true in the absence of sufficient oxygen, as shown by Syz [37] that if frogs are placed in an oxygen free medium, such as boiled water, oil, or nitrogen, the convulsant reaction to acid fuchsin is greatly increased. Kussmaul and Tenner,[12] in six non-epileptic subjects, produced convulsions in two by compressing the carotids. One of us (N) noted convulsions while compressing the carotids or vagi in a patient with paroxysmal tachycardia.

Popea and Eustatziu [38] have found that the inhalation of amyl nitrite averted or suppressed convulsions in 16 patients. The action of amyl nitrite as a rapid vasodilator is well recognized. It is reasonable to assume that this beneficial action came from its effect on the cerebral vessels, if as a result of vasoconstriction, an anoxemia had accompanied the seizure.

It appears that a vasomotor control of the cerebral vessels can be safely accepted.[30, 40, 41, 42] The completeness with which an area becomes blanched as a result of vasoconstriction can be seen in Raynaud's disease. The extremities effected change with marked suddenness. Deprived of blood, a localized anoxemia is produced. The association of Raynaud's disease with disturbances of cerebral vasomotor control has been noted.[43]

Cobb and Talbot [47] have shown that, in the brain as well as in the rest of the body, the proportion of open capillaries is constantly varying. Foerster [48] described a sequence of events during convulsions repeatedly seen by him on the operating table. First the brain becomes pale and sinks away from the skull; then it is suffused with blood and bulges greatly. Practically similar descriptions have been given by other surgeons, Kennedy [40] and Fay.[28] Hirschfelder [44] has shown that the retinal and the cerebral arteries constrict and dilate synchronously. A pallor of the fundi suggesting a vasoconstriction of the retinal vessels, associated with an epileptic attack, was described as far back as 1863 by Jackson [45] and in 1870 by Echiverria.[46] Jackson states that during the fit the veins become large and dark. These changes can be explained to our satisfaction only by a vasomotor disturbance. Lennox and Cobb [3] state that procedures which tend to cause seizures are in general those associated with constriction of the arteries, whereas many of the conditions which cause dilatation tend to inhibit seizures.

Probably nerve cells are least likely to initiate a convulsion when they are most abundantly supplied with oxygen and nutrient material. The beneficial effects of luminal may possibly be explained by its vasomotor action. Experimentally it has been shown that luminal sodium added to perfusion liquid in perfusion experiments on the head of a dog produced dilatation of the cerebral vessels as measured by the rapidity with which the fluid flowed from the brain.[50] Caffein, which produces dilatation of the cerebral vessels, has also been shown to have a beneficial effect on seizures (Karger,[51] Pethe[52] and Peritz[53]).

Whatever pathological condition exists in the central nervous system of an epileptic, it is permanent and probably progressive along with mental deterioration.[50] Why does the seizure come on at intervals with dramatic onset? We can but theorize as to the answer. It can best be explained to our satisfaction by a sudden vasoconstriction of the cerebral vessels which in turn produces firstly, anoxemia and secondly, edema. This edema may be quite sizable. Landis[54] has shown that in the presence of deficient oxygen supply, fluid escapes through the capillary walls of frogs at about four times the usual rate. When conditions favoring edema, as hydration and alkalosis, the factors which favor vasoconstriction, are present, the seizure is brought on more easily; but when acidosis and dehydration, the factors which favor vasodilatation, are present, the seizure is either prevented or modified.

We assume that in epilepsy there is a variable cerebro-spinal pathology which makes the individual more susceptible to seizures, and that vasoconstriction precipitates the attack. We do not know that a sudden vasoconstriction, to the extent seen in Raynaud's disease, takes place. The nature of the vasoconstriction may be not too different from that in the cerebral vessels of the non-epileptic. During and immediately after the seizures as a result of asphyxia and muscular contraction, there is a temporary acidosis. This helps to reverse the process as outlined to precipitate the convulsion and is a factor in depleting the brain tissue and producing normalcy.

We have then to produce convulsions: (1) A primary variable pathological lesion in the cerebrospinal system; (2) An adequate amount of body fluid; (3) A vasoconstriction as the precipitating factor.

We have pointed out that in diseases associated with dehydration and low blood volume, epilepsy is less frequent. For these reasons we were of the opinion that the epileptic should have a normal or increased blood volume. Lennox and Cobb [2] state that no work on the blood volume of epileptics has been done. Interest has been devoted heretofore to the interstitial and intracellular fluids of the body and the hydraulic pressure immediately surrounding the brain. We offer a preliminary report of our studies in the blood volume of epileptics.

The results of our studies in the blood volume of 78 untreated epileptics at interim, and their comparison with 42 controls, are shown in Fig. 1. There is a noticeable and unusually wide distribution of values among the epileptics. This corroborates the findings of Gamble who has shown that there is a disturbance of water metabolism in epileptics.[21] After considering all physiologic factors that may affect the volume of circulating fluids,[55] as posture, environmental temperature, exercise, age, sex and fat content of the body tissues, the comparison of epileptics with controls calls for explanation. Our group of controls consisted of 65 per cent men, and our patients 68 per cent men. Men generally have a higher blood volume than women but this slight difference in the sex distribution in the two groups could not explain satisfactorily the higher values in the epileptic group. The stepladder appearance of the lines representing the average blood volume by weight groups illustrates the parallel development of obesity with increase in body weight in the epileptic group. Another factor worthy of consideration is the inclusion of 8 young epileptics whose ages varied from 12 years to 16 years, and whose blood volumes were unusually high. However, eliminating these from the group, there remains a noticeably wide distribution of values, and an increase in the mean volume among epileptics as compared with controls. The blood volume in our 42 normal subjects ranged approximately 6 per cent lower than in Silbert's controls and 10 per cent lower than in Rowntree's controls. This may be explained to some extent by the fact that all our tests were done with the subjects in a sitting position, which, as shown by Thompson,[57, 58, 59] promotes concentration of plasma. The tests were done while fasting and with all subjects at approximately a basal level. A modification of Rowntree's [53] technique, as suggested by Silbert *et al.*[35] was used. Because

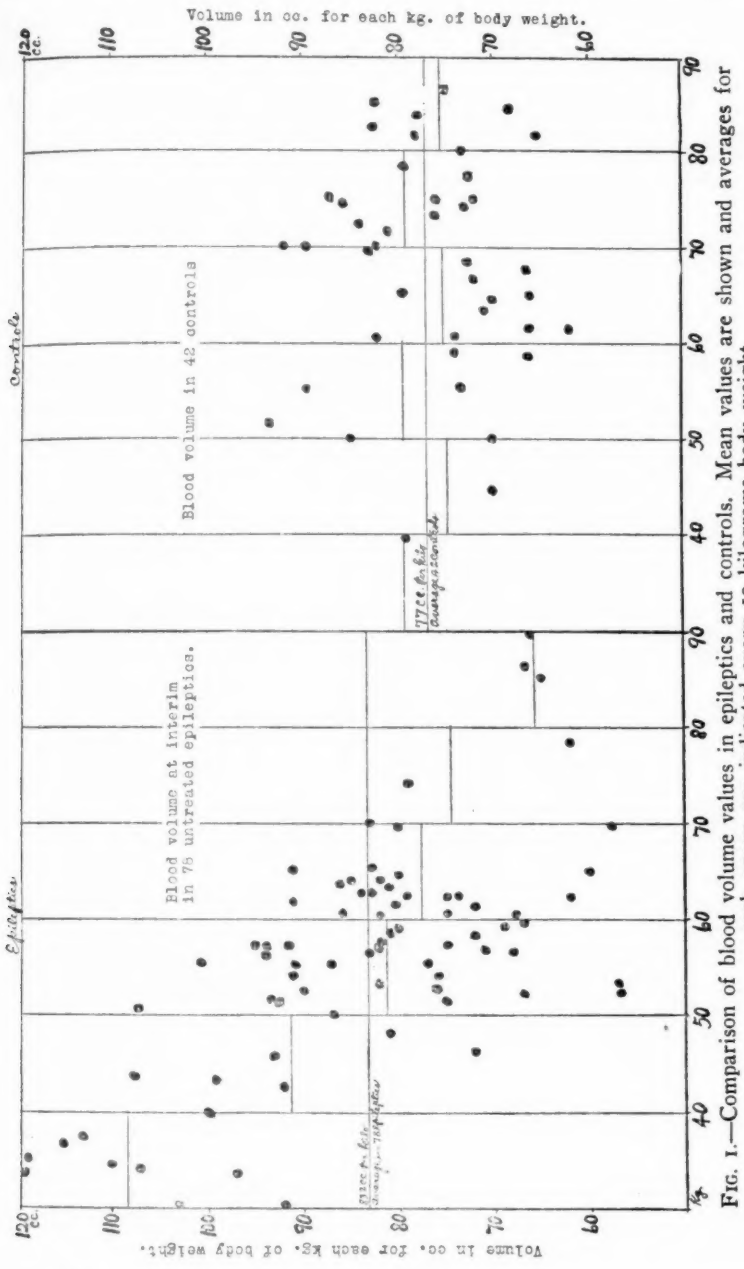

FIG. 1.—Comparison of blood volume values in epileptics and controls. Mean values are shown and averages for each group are indicated every 10 kilograms body weight.

of our lower averages among normals the relatively large group of controls (42) was obtained.

Fig. 2 is a graphic representation of the volume of cells, plasma, and blood in 42 normal subjects, 78 epileptics at interim, 13 epi-

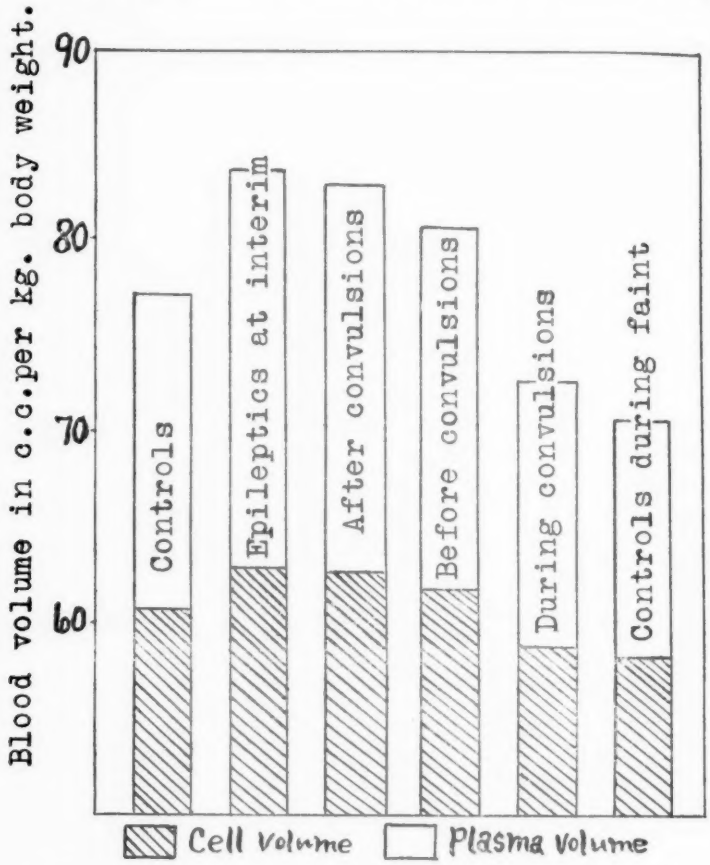

FIG. 2.—Graphic representation of mean values for volume of blood, plasma, and cells.

leptics who had had convulsions within a few hours before the test, three epileptics who had convulsions soon after the test, nine epileptics who either had convulsions during the test or who were in coma immediately after convulsions, and five controls who fainted between the time of injection of the dye and the time of

the second withdrawal of blood (5 min.). This figure again shows that the average blood volume per kilogram is higher among epileptics at interim than among the controls. This increase amounts to 8 per cent which indicates a definite trend. Table 1 is presented as a numerical explanation of Fig. 2. The average blood volumes among the groups who had convulsions before the test and after the test show only slight variations from the interim group and the changes are within the range of quantitative error. The group in which the test was done in immediate relation to the convulsive state ("during convulsions") shows a definite decrease in blood volume, as do the controls who fainted. The decrease in volume during seizures is an interesting, and not easily explained, phenomenon. The possibility of a mixing defect of the dye, due to

TABLE 1.

TABULATION OF MEAN VALUES OF CELLS, PLASMA, AND BLOOD VOLUME OF THE VARIOUS GROUPS.

	Cells per cent by hematocrit.	Plasma per cent by hematocrit.	Blood volume in c. c. per kg.
42 controls	39.7	60.3	77.
78 epileptics at interim	39.2	60.8	83.2
13 epileptics after convulsions	38.3	61.7	82.8
3 epileptics before convulsions	39.4	60.6	80.3
9 epileptics during convulsions	39.5	60.5	72.5
5 controls during faint	39.4	60.6	70.5

retardation in circulation, must not be lost sight of in these cases, and also in the fainting state. In fainting we assume that there is marked splanchnic engorgement which may capture a variable amount of blood and mechanically remove the same from the circulation. This could give us a lower blood volume. The factor of anoxemia in fainting may further deplete the circulating blood because anoxemia can produce tissue edema as we have shown elsewhere.

The question of the mechanism of low blood volume during the convulsive state, we have not as yet answered to our satisfaction and we are not now prepared to discuss. Further work which we are carrying on in our laboratory may throw some light on this problem. It is our impression that this apparent reduction in quantity of circulating fluid may be dependent upon a vasomotor disturbance.

As may be seen in Fig. 3 the average plasma volume computed in c. c. for each square meter body surface is practically the same in the epileptic and control groups. The average plasma volume

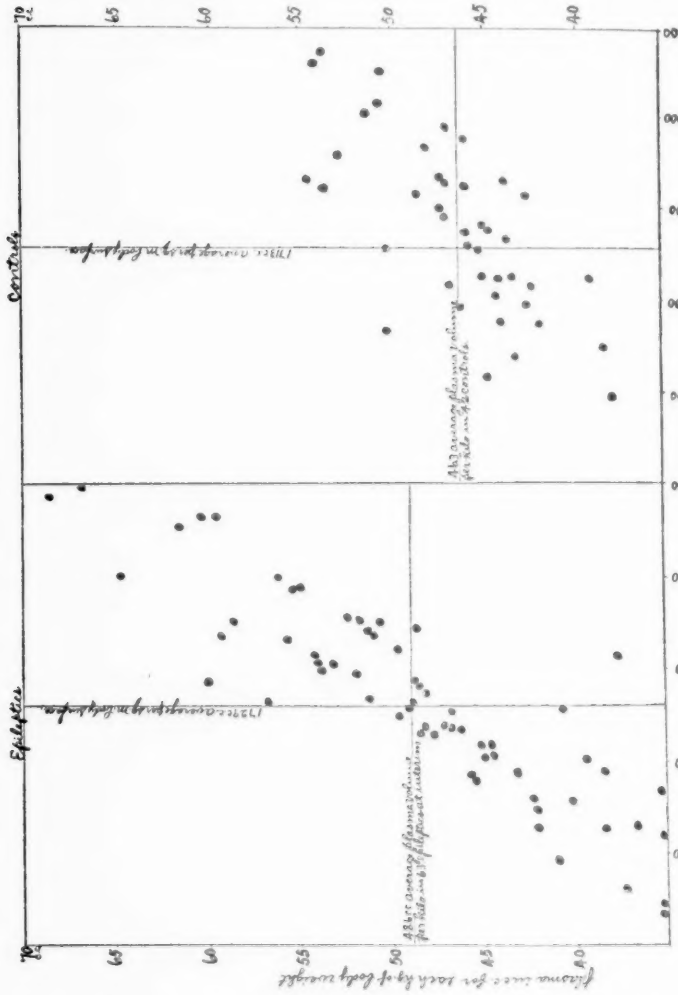

FIG. 3.—Comparison of values in epileptics and controls showing plasma volume for each kilogram of body weight and for each square meter of body surface.

in c. c. for each kilogram of body weight is slightly higher in the epileptic group (5 per cent). Similar to the distribution of blood volume values shown in Fig. 1, there is a wide distribution of plasma volume values in the epileptic group. This may be par-

tially accounted for by a wider variation in the weight of patients as compared with the weight of controls, assuming that the difference in weight is dependent, to some extent, upon the fat content of the tissues.

Table 2 shows the influence of ammonium nitrate therapy on the blood volume, plasma volume, CO_2 and plasma chlorides; and the clinical results are tabulated. Although there is a slight decrease in the blood and plasma volumes after ammonium nitrate, as reflected in the total averages, this change is easily within the range of error. The only definite change shown is a decrease in the plasma CO_2 following treatment. The plasma chlorides, while not estimated before treatment, appear to be normal after treatment. Clinically we did not observe any tendency to successions of seizures during the course of treatment as was observed by Lennox and Cobb; however, more patients showed an increase in frequency of seizures than a decrease. In the six patients who showed improvement the results of laboratory analysis showed no trend, nor is there any explanation suggested for those who had more seizures while on treatment. The weight in the entire group remained unchanged during treatment.

SUMMARY.

1. We have attempted to show that the cerebrospinal pathology in epilepsy is variable.

2. Dehydration is a factor in common in methods of treatment which modify the seizures in epilepsy.

3. The incidence of epilepsy is low in diseases in which there is present a low blood volume or a tendency to dehydration.

4. Alkalosis, edema, and decreased oxygen tension, conditions favoring convulsions are closely inter-related, as are acidosis, tissue dehydration and increased oxygen tension, conditions which decrease seizures.

5. A disturbance in the vasomotor control of the cerebral vessels is suggested as the precipitating factor of the epileptic convulsion.

6. A trend towards higher blood volume values was found in 78 epileptics as compared with 42 controls.

7. A low blood volume was found during convulsive seizures and during fainting attacks.

8. Acidosis, as is produced by ammonium nitrate therapy, is not sufficient to control seizures.

TABLE 2.

RESULTS OF TREATMENT WITH AMMONIUM NITRATE 90 GR. (6 GM.) PER DAY.

Case No.	NH_4NO_3 treat. No. Days	Blood volume per kilo.		Blood volume per sq. meter body surface.		Plasma volume per kilo.		Plasma volume per sq. meter body surface.		Plasma CO_2.		Plasma chlorides. After	Clinical results. After
		Before	After	Before	After	Before	After	Before	After	Before	After		
1	18	83.5	82.1	3067	2987	51	51	1871	1881	65.5	49.4	564	Unch.*
2	10	95	90.2	3437	3174	59.4	56.8	2138	2000	71	70	552	Unch.
	41	95	91.7	3437	3321	59.4	54.2	2138	1963	71	42	586	Unimp.
3	8	65	66.5	2848	2887	35.3	35.1	1538	1525	76	..	616	Unch.
	47	65	66.3	2848	2943	35.3	36.4	1538	1616	76	49	583	Unimp.
4	10	93	88	2840	2702	56.6	52.8	1732	1621	71.1	..	590	Unimp.
	44	93	92.2	2840	2970	56.6	57.1	1732	1843	71.1	42	593	Unch.
5	10	74.5	81	2579	2789	44.7	47.7	1547	1647	51.4	..	552	Unimp.
	44	74.5	81.8	2579	2829	44.7	48.1	1547	1666	51.4	58.9	588	Imp.
6	10	90	80.4	3061	2754	53.1	53.1	1866	1818	78.8	69	574	Unimp.
	42	90	73.4	3061	2533	51.3	45.1	1866	1558	78.8	48.5	577	Unch.
7	10	74	82.4	2576	2907	42.4	49.4	1520	1744	67	67	561	Unch.
	44	74	80.7	2576	2874	42.4	47.6	1520	1695	67	59.9	550	Unch.
8	10	107	81.1	3583	2845	66.9	52.9	2221	1790	67.3	..	611	Unimp.
	37	107	100	3583	3397	66.9	60.3	2221	2039	67.3	37	574	Unimp.
9	10	70.4	76.8	2637	2776	44.7	50	1635	1795	63.4	62.6	575	Imp.
	44	70.4	82.4	2637	2859	44.7	52.7	1635	1830	63.4	44.7	578	Imp.
10	10	90	88	2916	2997	45	51.9	1603	1768	61.3	71.8	525	Unimp.
	41	90	85.7	2916	2814	45	50.6	1603	1666	61.3	48.6	592	Unimp.
11	10	66.7	71.5	2908	2980	40.7	39.9	1716	1666	64.3	56	565	Unimp.
	44	66.7	80.4	2908	3388	40.7	44	1716	1853	64.3	53.2	576	Unch.
12	10	74	77.8	2698	2823	48.2	48.5	1755	1764	52.4	53.2	525	Unch.
	44	74	72.4	2698	2588	48.2	46.3	1755	1656	52.4	43.9	565	Unch.
13	10	75	73.2	2606	2555	42.2	41.6	1459	1456	67.2	76.8	561	Unch.
	44	75	80	2606	2880	42.2	46.9	1459	1686	67.2	64.5	560	Unimp.
	53	75	71.5	2606	2580	42.2	42.9	1459	1548	67.2	50.4	578	Unimp.
14	46	87	73.9	2902	2460	53.9	44	1799	1466	53	59.8	612	Imp.
15	30	75	57.5	3156	2133	49.6	39.6	1840	1471	55.1	49.4	594	Unch.
16	20	100	92	2941	2755	60	60	1765	1809	51.3	48.5	620	Unch.
17	18	62	62	2587	2535	38.4	37.3	1604	1521	49.4	49.4	562	Imp.
18	31	80	90	2907	3241	48.8	53.8	1770	1935	75.8	48.5	606	Unimp.
19	31	84.7	65	3035	2351	54.2	38	1943	1363	64	43.8	621	Unch.
20	35	91	93	3174	3180	57.4	56.2	1979	1920	60	48.5	576	Unch.
21	35	80	70.4	2897	2482	48.9	41.2	1767	1452	63	46.6	585	Unch.
22	35	80	85	3166	3277	54.6	40.6	1930	1910	62	50.4	583	Imp.
23	10	119	88	....	....	74.2	57.9	....	....	57.9	..	575	Unimp.
	35	119	101	....	....	74.2	61.4	....	....	57.9	50.8	501	Unimp.

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DISCUSSION.

Dr. S. B. Wortis (New York City).—I want to express my thanks to Doctors Hodskins, Guthrie and Naurison for the preceding paper, which represents an excellent review of the literature and a good amount of hard work at the Monson State Hospital.

That edema predisposes to convulsive seizures is quite evident to all of us, but that edema is the primary cause associated with all convulsive seizures is questionable.

At Bellevue Hospital I have tried giving intravenous hypertonic glucose to several epileptics. In some cases it helps; in others it does not.

We have tried giving amyl nitrite to some of our epileptic patients, either as amyl nitrite or sodium nitrite in daily doses sufficient to cause dilatation of the peripheral vessels. It helps some few—but not all.

In the laboratory, we have done cervical sympathectomy on animals—the results of which I reported this morning.

We have also used ergotoxin, which is a drug said to paralyze the sympathetic nerve endings. Giving an animal ergotoxin in large doses causes the animal to develop many evidences of sympathetic nervous system paralysis. But in all these cats we are able to induce convulsions by the use of camphor by vein.

I am sure all of you here have examined or seen the brains of persons having uremic convulsions. Not all of these brains are "wet," many appear dry to observation, both gross and microscopic.

Convulsive seizures have been known to be relieved following coincidental surgical operations. We have attempted (and are at present continuing this observation, on the Bellevue Hospital Neurological Service) giving epileptic patients ether anesthesia to surgical depth for one hour—without performing any surgical operations on these people—to see whether the effects of the anesthetic can diminish the number of convulsive seizures which will occur over a subsequent uniform time period. We find that 5 to 7 days after these persons have had their anesthetic, they go on having convulsions, with the same frequency as before.

In regard to convulsions coming on following brain trauma associated with blood in the subarachnoid space, I believe we owe thanks to the observations that were made by Dr. Bagley of Johns Hopkins. Doctor Bagley showed, four years ago that when he introduced blood into the subarachnoid space in animals in small repeated doses, these animals developed spontaneous convulsions, and subsequently showed hyperplastic changes in their meninges often with associated secondary changes in the cerebral cortex.

I want to make one other comment—and I think Dr. Pike will confirm this observation from his laboratories—that animals having infections of any sort, whether it be a cold or pneumonia or severe diarrhea, are much less subject to convulsive seizures by standard amounts of the convulsant drugs we use.

This confirms clinical work which has been reported by Dr. Guthrie on various types of infections causing a diminished number of convulsive seizures in epileptic patients.

I want to take this opportunity once more to say that from our work in the Laboratory of Experimental Neurology at Bellevue Hospital we feel that vasospasm is merely an expression of the fit. I have no doubt that vasospasm can cause convulsions in many cases, but that it is the primary pathology in all cases is questionable.

I feel the same way in regard to brain edema.

DR. M. S. GREGORY (Oklahoma City, Okla.).—We hear in the West a great deal about allergy and epileptic seizures. I wonder if Dr. Naurison will kindly touch upon that subject for a moment.

DR. F. H. PIKE (New York City).—I have the usual questions—whether in a child that has never had a convulsion there is an increase in the blood volume before the first convulsion appears. It is a somewhat puzzling question.

Then again, I have noted the very wide range of variation in the blood volume of epileptics. Some of them have a much lower blood volume than others. What is the severity of the convulsions in those patients with the lowest blood volume, as compared with the average case; and does this blood volume change with the age? Is it a condition which gets worse with the progress of the disease?

DR. FRED CURRIER (Grand Rapids, Mich.).—I would like to ask one question. You have spoken here of the beneficial effect of infection on convulsive seizures, but how do you harmonize that statement with the fact that in certain diseases, like measles and whooping cough and scarlet fever, we get the first appearance of convulsions?

DR. S. B. WORTIS (New York).—That is a question we usually expect, Dr. Currier, and this is the answer we usually give, and I think Dr. Pike will confirm it.

The time one sees convulsive seizures in children complicating the exanthemata or complicating any systemic infection, is very early in the disease, before the temperature develops. Convulsions are related to toxemia we feel, rather than to fever.

DR. F. H. PIKE (New York City).—On that point, the type of infection seems to have some relation to the effect. Dr. Hodskins mentions that some of his patients had convulsions which ceased during a lobar pneumonia, but that they continued unaffected during typhoid.

I don't suppose the surgical results in animals were any more nearly 100 per cent perfect than in the hospital cases. Cats sometimes have some bacillus concealed in the skin which it is very difficult to eradicate. We occasionally get infections with foamy pus under pressure.

In one animal of this sort we gave the minimum convulsive dose of absinth before opening this abscess and afterward. Simply loosening some stitches

and letting this foamy pus escape, without doing anything else whatever to the animal, raised that convulsive dose considerably. I think the type of infection makes a difference, and some of the other incidental effects seem to bear out this view.

I raised the question two or three years ago as to whether the onset of malaria, which must occur in some of our institutions, had any effect on the frequency of convulsions. I have not yet received an answer to that. There must be clinical observations somewhere on that point. If there are, I do not know about them.

DR. MARY E. POGUE (Chicago).—I should like to ask if there is any special type of epilepsy on which psychotherapy has been tried.

DR. JAMES Z. NAURISON (Springfield, Mass.).—I should like to reread three or four lines of this paper, because to some extent that must be the basis for the discussion:

"We are confronted with the idea that individual variations in the manner of reacting to environment is characteristic of living material and in the presence of a given stimulus only certain individuals may react with convulsions."

We have attempted to show that there is no fixed pathological condition, and we do not propose to assume such a thing. There must be a variation in living individuals, living material, the reaction of living cells, and if someone will explain to our satisfaction why certain diseases happen to certain people, we can explain other things in turn.

It has been shown, for example, by Dr. Draper of the Rockefeller Institute and by Dr. Freeman of Washington, that individuals with certain anatomical characteristics are prone to certain diseases. Although they live the same life and are in the same environment as others, they develop such diseases more readily. In general medicine one can foretell that certain types of individuals may perhaps die of nephritic disturbances and heart disturbances, and that others may be more liable to develop tuberculosis. Why certain things happen, and why certain things do not happen, is one of the mysteries of medicine.

I should like to ask Dr. Wortis whether in the use of ergo-toxin it took the same amount of camphor monobromide to produce the convulsion, or a larger amount.

The fact seems to be that certain individuals are especially liable to, and will have convulsions; but that these individuals will have convulsions less often under certain circumstances, for example if their brain cells are more adequately supplied with nutrient material, especially oxygen. We are of the opinion that these at least, are control factors. If, to produce a convulsion the animal which has been given ergo-toxin requires a smaller amount, or the same amount, of camphor monobromide, we will admit we are all wrong; but, if it takes a larger amount of camphor monobromide to produce convulsions, I think we may still stand our ground.

With reference to the anesthetic, I brought out in the paper that the surgical operation produces a favorable change. You will please remember that when an abdomen is opened and the intestines and the abdomial contents are handled, a certain amount of temporary paralysis takes place. Subsequently there is a certain amount of vomiting, a certain amount of fever reaction, perhaps a protein reaction; blood or other material has to be reabsorbed.

As factors additional to the anesthetic we have—(1) the temporary paralysis of the intestinal tract, (2) vomiting, which favors dehydration, (3) fever, which also produces a certain amount of dehydration. There are present then several factors to produce starvation and dehydration. Where one has simply administered an anesthetic over a period of time all of these factors are not present and this may explain the difference of effects in reducing seizures.

Dr. Wortis brought up the question of trauma. We discussed that in our paper and feel it is an important factor, but it doesn't seem to be the factor for all cases. We have to go back again to the idea that certain individuals with the same trauma, as far as we can tell, will have a convulsion and others not susceptible to convulsions, but with equal trauma, will not have convulsions.

We still feel that the vasomotor disturbances give us our most logical answer as the precipitating agent for the seizure. If the vasomotor upset comes subsequent to the onset of the convulsion, we still have to ask the question: What brings on the original convulsion? Certainly the pathological change in epilepsy is there permanently and it is progressive. The brain of the old epileptic is almost always a different type of brain from the brain of the comparatively new epileptic. The old epileptic brain has a more shrunken appearance, we think. The tendency for convulsive siezures is there all the time. Something precipitates the convulsion. What that something is can best be answered to our satisfaction by a vasomotor disturbance.

Dr. Gregory asked about allergy. We think there isn't a great deal of difference between allergy and a temporary, localized vasomotor disturbance. We do not know enough about it to discuss it. Therefore, we did not include it in the paper.

We do feel there is some sort of similarity between migraine and epilepsy. There has been quite a good deal written on the subject. That the association between the two is rather close, is suggested by the study of etiological factors and inheritance in epilepsy at Monson Hospital, showing that the number of epileptics with parents who have had migraine is quite high. If my memory does not fail me, Ely of Des Moines, Iowa gives ancestral incidence of 60 per cent in all those who have congenital and inherited epilepsy. That may not be the entire cause, by any means, but there appears to be something in common between the two diseases. Certainly in migraine one can control the attacks with a ketogenic diet to a very fine degree, even if one allows foods to which the patients are sensitive. An excellent paper on migraine was presented in Baltimore last March before the American College of Physicians.

Dr. Pike spoke about infections being a factor in the elimination of the convulsion. We feel that infection alone is not a factor, but that when infection is associated with temperature it becomes a factor. We have seen infections in which the convulsions go on uninterrupted. A child who previously may have had a series of convulsions, develops a fever, and subsequently there is a remission of seizures. But this remission is not a definite thing. The remission is shorter in some cases and longer in others, and it is due we think to the conditions associated with fever.

Dr. Pike asked about the increase in the blood volume before convulsions appear. We don't know. We have just started this work, and we have lots of things to learn about it. Our lowest blood volumes have been with short, fat women; their obesity causes low blood volume. What we expect to do is to check those who have a low blood volume against the standard tables that have been proposed by Rowntree, deducting our ten per cent difference from his numbers, and then perhaps we can answer that question a little better.

On the question of typhoid therapy, I mentioned that nonspecific protein therapy had been proposed as long as ten years ago, by Milner and others. The question becomes one of fever and its possible beneficial effects. Whether the nonspecific protein therapy or typhoid germs will give the same benefit as malaria, may depend upon the extent of the fever. There may or may not be a beneficial result according to the water loss produced.

Dr. S. Bernard Wortis (New York City).—I would like to say that ergotoxin or amyl nitrate, when given in doses, sufficient to ensure the desired pharmacological reaction in animals; does not alter the amount of camphor required to induce experimental convulsions.

Work reported by Wolff on cerebral circulation, showed that when the cervical sympathetic had been cut, increased vascularity of the related pial area resulted. Related work on animals by Meagher and Ingraham in 1928—wherein, they tied off the carotid vessels on one side and stimulated the cervical sympathetic on the other side, but were not able to produce convulsions—is worthy of mention here.

II. EPILEPTIC REACTIONS IN THE NEGRO RACE.*

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I. INTRODUCTION.

This paper is the second in a series of expositions of mental reactions in the negro. The first, which was a general review of the field of comparative psychiatry as related to the negro, indicating several important differences in their psychobiologic activities, was read recently before the Washington Society for Nervous and Mental Diseases, and is as yet unpublished.

In the course of the existence of St. Elizabeths Hospital about 255 negroes and negroid epileptics have been carried on their rolls. Of these 160 were selected for the study, as the records made in the early days were not sufficiently inclusive or detailed to warrant their utilization in present day investigations. No consistent attempt was made to determine the degree of white blood admixture in these types.

Davenport (1923) in his studies on the geographic and racial distribution of epilepsy emphasized that the negroes in the United States are largely mulattoes who have from contact acquired the vices of the whites, and that these mulattoes show great predominance in epilepsy over whites, confirming the hypothesis that human hybrids are disharmonious and exhibit predominance of nervous defects over the "purer" races. Therefore, "according to this theory miscegenation increases the number of new centers of epilepsy while imbreeding tends to increase the proportion of epileptics in the descendents of one of them."

From the material gained from adult men of both colors examined in the 1917-18 war draft, Davenport found that in those areas of the southern states which are inhabited by 45 per cent or

* Read at the eighty-seventh annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Toronto, Canada, June 1, 1931.

more negroes there was a slightly lower rate for epilepsy (5.13 per cent) than for prevailing white agricultural districts (5.65 per cent) or for "mountain whites" (6.28 per cent). Hirsch (1886) concluded that epilepsy appeared in the same form and with the same frequency among black races of the west coast of Africa as in Europe and other countries, and Chassanial (1865) found epilepsy in Senegal much the same as in Europe. Bonius (1882) from the same place considered epilepsy to be of frequent occurrence. Clarke (1860) of the Gold Coast remarked in describing diseases of this district that epilepsy was occasionally brought to his notice, and Mooney of the Gold Coast Asylum at Accra reported 13 cases of epilepsy in 200 cases of mental disorder in the West African natives.

Warnack (1924) in his long experience (28 years) with mental disorders in Egypt among Egyptian blacks found epileptic insanity to be as common as in England. The running "amok" so common among primitive peoples and which Kraepelin tended to interpret as an epileptic episode is considered by Van Loon (1927) to be the result of an infectious etiological process, usually malaria, typhoid, or influenza, acting upon a race in which the emotivity is intense and suggestibility is high, almost without resistance to sudden emotions thus causing panic.

Green (1914) in a large group (5410) of white and negro mental patients in the United States found that epileptic psychoses occurred with about equal frequency in both races (8.05 per cent whites—8.4 per cent negro). Bostock (1924) in writing of mental disorder in the Australian aboriginal reported some form of epilepsy in 30 per cent of cases as compared with 10 per cent among the whites admitted to the same institution. (Mental Hospital—Callan Park, N. S. Wales.) The natives exhibited the same type of epileptic reactions as seen in the whites, and seemed to have similar types of aura, *e. g.*, one patient felt "sullen" in the stomach as descriptive of an epigastric pre-convulsive sensation. This remarkable increase in epilepsy on the "fringe of civilization" led him to remark upon the possibilities in the protein sensitization theory to account for the phenomenon and to consider epilepsy in the light of a possible factor in racial extinction.

Bailey (1922) in his neuropsychiatric experience during the World War found 13.3 per cent epileptics among 8406 classified

neuropsychiatric cases in negroes, as compared with 8.6 per cent in the general United States average based on 69,394 cases.

Pollock and Furbish in their statistical survey of epileptics in the institutions of the United States found that of the 8777 patients institutionalized for whom schedules were obtained, 8601 or 98 per cent were white, 173 or 2 per cent negro and 3 were Indians. The rate of resident whites per 100,000 of white population was 9.1 and correspondingly for negroes 1.7. These authors remark that "The figures do not indicate that the rate of epilepsy is lower among negroes, but that a smaller proportion of negro epileptics are cared for in institutions. Such statistics are perhaps sufficient to give a general idea of the comparative situation in numbers.

II. TYPES OF CASES.

After making a general survey of our 160 cases it was thought advisable to group them for study according to the outstanding clinical picture gained from the course of the disorder. Cases of convulsions associated with clear cut organic brain disorders (symptomatic epilepsy) such as general paresis, brain tumor, advanced cerebral arteriosclerosis, toxemias, etc., were not included in this study, but only those that were more obscure or idiopathic in expression and also those with some degree of original mental deficiency were selected.

The selected cases fell fairly readily into the classification shown in the following table:

TABLE I.

CASES OF NEGRO EPILEPTICS.

Groups.	Living. M	Living. F	Discharged. M	Discharged. F	Dead. M	Dead. F	Total.
I. Epilepsy with confused states	1	12	12	11	21	13	70
II. Epilepsy with progressive deterioration	6	12	6	7	12	9	52
III. Epilepsy with mental deficiency	0	6	5	0	10	6	27
IV. Epilepsy with schizophrenia	1	0	4	1	0	1	7
V. Epilepsy with organic brain disease	0	0	0	0	3	1	4
Totals	8	30	27	19	46	30	160
	38		46		76		

III. OBSERVATIONS ON GROUPS.

A. Epilepsy with States of Confusion.

Of the 70 cases, 34 were males and 36 females, concerning which we shall now proceed in detail with each sex.

MALES.

1. *Age* on admission varied so as to be of no importance in this particular study.

2. *Social condition* showed the usual negro variations in marriages, illegitimacies, and promiscuities.

3. *Education* was less than eighth grade throughout.

4. *Heredity*.—The usual difficulty in obtaining reliable family histories from negroes was encountered but some important instances were recorded:

	Cases.
Parent epileptic	2
Parent psychotic	1
Parent alcoholic	3
Parent apoplectic	1
Parent tuberculous	1
Parent syphilitic	1
Sibling epileptic	1
Sibling tuberculous	2
Near relative psychotic	1
Near relative epileptic	1
Total	14

5. *Alcoholism*.—In 13 cases there was a history of excessive alcoholism previous to the onset of the epilepsy or during its course or both. There was no history of drug habits in any of the cases.

Personality.—The economic, social and sexual prepsychotic adjustment varied greatly from none at all to fairly successful adaptation; however, actual misdemeanors and arrests were comparatively rare. The much mooted "epileptic personality" of Clarke and others was determined to have been distinctly present in the preconvulsive lives of four patients.

7. *Convulsions*.—The age of onset, type of attack, frequency of convulsions, auræ, equivalents, etc., have been satistically consid-

ered in another paper; here it will suffice to say that all patients included in the study were subject to major convulsive attacks, the onset of which varied from early infancy to comparatively late in life. Among the 34 cases in this particular group only two began in early infancy and two at puberty. One case had the typical "running fits" as well as major convulsions, and one had a migraine-epileptic combination.

8. *Outstanding Mental Symptoms.*—

(a) *Confusion:* Marked confusion either before or following the major convulsions was the outstanding characteristic of the group.

(b) *Metaphysical Delirium:* Of the 34 patients 15 had a metaphysical delirium during their confusion, characterized by excited, destructive, often homicidal behavior, with odd mystical ideas based on hallucinations, frequently of religious import, but mixed with ideas of death, blood and rebirth. The following case extracts will serve to illustrate the reaction. One patient said he had visited both heaven and hell and had heard a serpent telling him that he would never get to heaven without going through hell. His conversation was confused, particularly after attacks, as he had post-epileptic confusion for a few hours after each spell, during which he complained of "indigestion" and "liver swells up and gets costic." He had periods of confusion and excitement as epileptic equivalents, during which he was dangerous. In the intervals he was a good worker, quite cooperative and showed no psychotic features. His memory was good. He had elaborated hypnagogic hallucinations. He heard and saw God, became excited and broke down doors. He maintained that he had visual hallucinations at 11 years of age and at intervals thereafter until the present. He referred to his epileptic spells as choking spells. His stream of talk was over-productive and rambling.

His auditory hallucinations were vivid and weird (metaphysical delirium). Once he heard and saw the devil and believed he was in heaven and hell at the same time. Railroad ties were being made and cut off, laid over his chest and trains were put on them. Voices told him he would be shot in the head "about the woman I saw the doctor treating." . . . . "Something wrong with her—fire—I could see her whole 'innards' and it looked like a map in geography and I could hear the whole world sing in a whirlwind. Everybody

was in heaven, but the devil." He dreamed one night he was in hell and " did not give a damn if I was at the bottom." One night he saw death, who told him of heaven and hell. Just in front of this vision he saw a lady lying down and a man standing, possibly a doctor, treating her nose. He could see right through her head and the brain was visible. The patient himself appeared to have blood on his hand and he heard a boy who was standing by him say, " Boy, you are going to die," but death told him that he would keep him alive. He then saw a white gentleman with long white hair.

The patient also made the following statements, " I always have fits about the time the moon changes, from increase to decrease of about full moon—I can tell when they are coming on by looking at the moon." He constantly complained of suffering from constipation. He broke a window and said God told him to do it and then stated, " When God or Christ tells me to do anything, I must do it, even if I die the next minute." Voices came from the inside and he heard them as a small child. The Lord speaks very distinctly and once showed him his grave and said that it was the starting place of heaven.

Another patient said, " They were going to make me imagine I was on fire and make me jump out of the window." He saw people with sheets on preparing for the burning up process. These first delusions came on following a spinal puncture. He begged not to be tortured too much and thought he was at the mercy of the physician. " Imagination makes me go through my life from birth until the end of time and back again." He begged for medicine to relieve this spell of imagination. He was usually confused and troubled about himself and got a telepathic message that the physician knew his early life and that the epilepsy really did not come from sun stroke. He did not know from whom the message came. Once he fell on the bed, saying that the building was coming down and for the physicians to run away to prevent being injured. Then he thought he was burning up and yelled, " God or the devil has nothing to do with it." (Later he expressed insight into this episode, and it was probably an epileptic equivalent.) Then he began to get messages from Christ and the President, simple messages like telling him to go to bed, etc. He talked about himself smilingly and later began irritating patients in a moderate way. He had to be

secluded whenever the power of God came over him. Twenty-four hours after a convulsion he made the following statement, " I feel as though I have a world here. (Pointing to heart and chest.) I have a live apple downstairs which has grown into trees and has come back to earth in human flesh. I have learned everything on earth. I feel as though I had the power and wisdom of Jesus Christ. I am God Almighty and have turned the world wrong side out. I am Jesus Christ on earth. I will walk through the Valley of Death and will bring the Shadow of Death. God Almighty has made me in his strength and I have made God in his." He became excited, violent and threatened to annihilate the earth. " I have millions of words and unlimited power. I can speak with the spirits of the grass, trees and sun. I was born before the world. I ate some fruit and trees came out of my mouth. I swallowed the seeds of trees. The seeds grew in the earth and I have transformed power from them." All of this is somewhat obscure, but he referred to the trees as his off-spring. These talked to him out of the trees. He might be sitting under a tree and the latter would converse with him on various subjects, history, scripture, etc. He could talk also to the waves of the sea. One day he got voices from the sun telling him of the diseases of the human body. He had 18 or 19 hundred trees scattered all over the world so that they could answer him whenever he called. The other night a voice came from the moon, spoke to him at the time a light came down and circled his head. He said they just jollied him, asking him how as was getting along and he supposed it was father (as he was closest to his blood).

(c) *Anxiety:* In seven instances there were special feelings of anxiety, fear of impending danger and definite fears of going to be killed for supposed crimes accompanied by the usual pleading for mercy and for help.

(d) *Sadism:* In 27 cases sadistic activities were outstanding and on occasions led to serious results, mostly destructive attacks on clothing and furniture; although perhaps acts of sadistic import were not recorded as such, but only attacks on other persons in the intervals between convulsions. Sadistic acts were frequently colored by religious ideas and sometimes motivated by such ideas.

(e) Extreme *relioiosity* with mystical ideas concerning the activities and influence of God and the Devil was prominent in 12 instances.

(f) *Paranoid* projections ranging from mild complaints against those in the immediate environment to organized systems of persecutory delusions were found in eight cases.

(g) *Hallucinations:* Olfactory and gustatory hallucinations were not found in this group; but auditory hallucinations principally, although not wholly, of religious content were found in 16 cases, and both auditory and visual hallucinations of similar nature in 15 cases.

(h) Actual *depressive content* was found in only one patient, which is in keeping with the low rate of depressions among the negroes as a race.

Suicidal gestures had occurred in two cases and one had indulged in some suicidal thoughts, but these patients were more than half white.

(i) A *catatonic reaction* was characteristic of the preconvulsive state of one patient.

(j) *Magical or superstitious interpretations in connection with convulsions,* were of very common occurrence. The fits were most frequently related to changes in the moon (" full moon "—" moon on the wane "—" new moon," etc.). One patient said that " blood smells and sights " caused the fits, another thought that a vision of " people with large teeth eating others " caused the spells, and several alleged that they were due to " spells or voodoos " put on them by ill disposed persons or malignant old women. For example one man knew that he had fainting spells and that some old woman had something to do with it. He thought this old woman had a grudge against his mother and had given him a dose of medicine which had knocked him out. The dose he took contained lizards' eggs which were so small he could not see them, nor did he recognize any peculiarity about the dose. These lizard eggs hatched and the lizards had been growing for some time. He stated that he could feel them now in his stomach or left arm. He thought some of these passed out through his abdominal wall. Once he saw these lizards reflected through his body in the shadow as caused by the sun. On one occasion he saw snakes as well as lizards bobbing up before him. States that he went to see this old woman to square up matters and she threw a cup of coffee in his face, and then he was entirely certain she had done it. These lizards are mostly yellow with

black spots, but their color is changeable. He declared that he sometimes had headaches before these attacks and that after an attack did not feel right for several days. He wanted to die and thought that he should commit suicide. He thought at times that he had left the world completely, sometimes was in heaven and sometimes was in hell. He saw his father and mother in heaven and saw the devil with hoofs and horns. Hell was full of ugly people who would run at him and annoy him. Sometimes he struck other patients because he thought they were making speeches about him. Sometimes blows were exchanged and the patient was not always the victor. Once he had a row with a patient who blackened both of his eyes and cut his head in several places. In June he had two severe convulsions which were the only ones he had had since admission. They were typical grand mal affairs with post-epileptic confusion.

9. *Physical and Laboratory Findings.*—

(a) *Serology* was negative throughout with the exception of one case with strongly positive Wassermann reaction in blood.

(b) *Neuro-somatic deterioration:* Syndromes (Hodskins) to be described later in this account were found in seven cases.

(c) *Other physical findings:*

	Case.
General syphilis	1
Cardiac hypertrophy	1
Gastric carcinoma	1
Feminine hair distribution	1

10. *Discharges.*—Of the 34 patients in the group 12 were discharged from the hospital, one as recovered (age 47), seven as improved, and four as unimproved.

11. *Deaths.*—Of the 34 patients in the group, 21 died while in the hospital.

(a) *Causes of death:*

	Cases.
1. Status epilepticus.	
(a) Pneumonia complications	8
(b) Cardiac failure	4
2. Asphyxiation	5
3. Lobar pneumonia	1
4. Acute cardiac dilatation	2
5. Gastric carcinoma	1

(b) *Autopsies* were performed on seven cases, revealing the following lesions having perhaps some special bearing on the disorder:

	Instances.
1. Hypoplastic external genitalia	2
2. Hypoplastic circulatory system	1
3. Mitral stenosis	3
4. Closed sella turcica	2
5. Generalized tuberculosis	1
6. Cerebral arteriosclerosis	1

(c) *Microscopical* examinations revealed one case in which the brain showed the typical lesions described by Spielmeyer, to be mentioned in more detail in the discussion at the end of this paper.

FEMALES.

1. *Age* on admission varied so as to be of no importance in the study.

2. *Social condition* showed the usual negro variations.

3. Formal *education* was less than eighth grade throughout.

4. *Heredity.*—

	Cases.
Grandparent—alcoholic and epileptic personality	2
Grandparent—psychotic	1
Parent—epileptic	5
Parent—psychotic	3
Parent—alcoholic	5
Parent—cancerous	1
Siblings—epileptic	2
Siblings—psychotic	3
Siblings—tuberculous	1
Total	23

5. *Alcoholism.*—In seven cases there was a history of excessive alcoholism previous to the onset of the epilepsy or during its course or both. No history of other drug habits in this group.

6. *Personality.*—The economic, social and sexual prepsychotic adjustment resembled that for the males with history of misdeameanors comparatively rare. The "epileptic personality" previous to onset of convulsions had been present in four instances.

7. *Convulsions.*—All patients were affected with grand mal convulsions, the onset of which ranged from early infancy to com-

paratively late in life. Among the 36 patients, 11 began in infancy, eight with onset of menstruation, one with pregnancy and three with menopause. Thus 12 or 13 of the cases seemed to be related to the sexual epochs and functions.

8. *Outstanding Mental Symptoms.*—

(a) *Confusion:* Outstanding confusion either before or following the major convulsions was the principal characteristic of the group.

(b) *Metaphysical delirium:* Of the 36 patients nine had a typical metaphysical delirium as described for the males.

(c) *Anxiety:* In only two cases were there special feelings of anxiety, fear, depersonalization and hypochondriacal ruminations.

(d) *Sadism:* In 25 of the 36 cases sadistic activities were in the foreground, and often accompanied by religious and erotic components, an irritable, antagonistic disposition being present almost constantly.

(e) Extreme *religiosity* with mystical, superstitious interpretations and attitudes was present in five instances.

(f) *Paranoid* projections with various delusions including the prominent ones of poisoning were noted in nine cases.

(g) *Hallucinations:* Auditory experiences were present in ten patients and both auditory and visual hallucinations in six patients.

(h) A *depressive content* was found in only one patient and there were no recorded suicidal tendencies in the group.

(i) No *catatonic* states were observed.

(j) The remarks made before on magical and superstitious interpretations of events apply as well to this group of females.

9. *Physical and Laboratory Findings.*—

Spinal fluid serology was negative throughout, but the Wassermann on the blood was positive in five cases. X-rays of the skull showed a closed eroded sella turcica in five cases.

(b) *Neuro-somatic deterioration syndromes* and other evidences of " shattering " of the nervous system were found in eight cases.

(c) *Other physical findings:*

	Cases.
General syphilis	5
Heart lesions	1
Hemiplegia	1

10. *Discharges.*—Of the 36 patients in the group, 11 were discharged from the hospital, six as improved and five as unimproved.

11. *Deaths.*—Of the 36 patients 13 died while in the hospital.

(a) *Causes of death:*

	Cases.
1. Status epilepticus.	
(a) Pneumonia complications	4
(b) Cardiac failure	2
2. Asphyxiation (convulsion)	1
3. Toxemia from burns	1
4. Streptococcic infection	1
5. Disseminated tuberculosis	3

(b) *Autopsies* were performed on six cases revealing the following lesions:

	Instances.
1. Cardiac hypertrophy	1
2. Disseminated cerebral sclerosis, particularly of Ammon's horn	3
3. No lesions in brain	2

(c) *Microscopical* examinations of one brain showed the lesions described by Spielmeyer.

B. Epilepsy with Progressive Deterioration.

Of the 52 cases, 24 were males and 28 females, concerning which we shall proceed in detail as before.

MALES.

1. *Age* on admission varied as usual.
2. *Social condition* showed usual negro variations.
3. *Formal education* universally below eighth grade.
4. *Heredity:*

	Cases.
Grandparent, psychotic	2
Parent, epileptic	2
Both parents, epileptic	1
Parent satistic (epileptic personality) (one executed for murder)	2
Parent, alcoholic	3
Siblings epileptic	3
Five siblings, epileptic	1
Siblings, mentally deficient	1
Siblings, tuberculous	2
Others psychotic	1
	18

5. In nine cases there was a history of excessive alcoholism previous to the onset of the epilepsy or during its course, or both. No history of other drug habits in the group.

6. The economic, social and sexual prepsychotic adjustments resembled the former group with the exception that there was one homicide in this group whose father had been executed for murder, and had shown the "epileptic personality" type of character. The "epileptic personality" was present according to the histories of this group in two instances.

7. *Convulsions.*—All patients were affected with grand mal convulsions. Among the 24 patients, only two convulsive states began in infancy, the remainder were distributed through early adult life.

8. *Outstanding Mental Symptoms.*—

(a) *Deterioration:* Progressive deterioration in the intellectual and emotional spheres was the outstanding expression of the entire group. In only three cases was there any prolonged period of marked confusion preceding or following the convulsions.

(b) *Metaphysical delirium:* This reaction was seen in only three patients and was associated with the confused cases mentioned above. As an example of one of these "metaphysical" states the following case extract is offered. The patient's convulsions were followed by a deep stupor for an hour and then confusion for an hour or so. He had seven convulsions in eight days, during which he was noisy, singing and praying. He then spoke of his many previous heterosexual experiences. One night he thought he was in Jerusalem with his wife and had walked through fire the same as John. He showed one of his burned hands as proof of this. He indulged in numerous fits and had one epileptic ecstacy or hysterical episode, in which he remained for several hours. Said he was Christ during this time and was happy that his future was assured. He had many outbreaks of violence, but between convulsions was pleasant and agreeable in every way. He talked in a muffled, thick manner. He helped with the ward work, but had strong aversions to work. His head was often cut badly as a result of accidents during the epileptic spells. He seemed to have vague hallucinations of a religious nature most of the time. Heard his dead mother's voice and said he was going to Jerusalem. Once he had a peculiar tantrum and said his father

had been to heaven, but it was not time for him to go. Said he had passed through heaven and back again during a post-epileptic confusion. He heard singing, bands playing and saw flowers growing there. Said he must not talk so loud as it would be disturbing them, as he could still hear them. During some convulsive periods he was not entirely unconscious. Once he had a prodromal headache and went directly into an ecstacy. He felt elevated, high up in space, but did not know just where. He felt as if he were flying about in space or in heaven. He saw flowers of silver and gold, which brightened everything before him. He could not say whether his eyes remained open or closed, but he saw grand colored visions—silver and golden moons and stars, kings in bright carriages, and saw his Master, not face to face, but clothed in fair raiment of brilliant colors, sparkling with gold and silver. He had seen his mother and dead relatives in a similar manner. As a rule he saw no visions at these times. During the visions, legions of people flew about in beautifully lighted fields. They looked like angels. He was in rapturous ecstatic joy. In his earlier spells of this type he was overawed. Now he has become used to the experience. After the visions had passed he felt happy and cool in the head. He was then excited and over-talkative all of his conversation being of a religious trend. He often said, "Almight Lord's son."

Mild fear states with acute projections were seen periodically in three patients.

(d) *Sadism:* In 15 of the 24 cases sadistic activities were prominent, in this respect being in keeping with the findings for the group characterized by confused states. One man had committed a murder for which he was doing a life sentence.

(e) Extreme *religiosity* was noted in only two cases.

(f) *Paranoid* projections with delusions of persecution were seen in only three cases.

(g) *Hallucinations:* Auditory hallucinations were noted in three patients and were accompanied by visual experiences in two instances. Other types of hallucinations were not encountered.

(h) A definite depression was noted in two patients, and in one of these there were two suicidal attempts which seemed to be genuine. (One attempt at hanging and one shooting episode.)

(i) No catatonic states were observed.

(j) Previous remarks on magic and superstition obtain here as well.

9. *Physical and Laboratory Findings.*—

(a) All serology was negative throughout the group.

(b) *Neurosomatic deterioration syndromes* and isolated evidences of nervous system involvement, such as hypertonicity of muscles, choreoathetoid movements of upper extremities and marked speech defects were found in 10 cases.

(c) *Other physical findings:*

	Cases.
General syphilis	1
Pulmonary tuberculosis	1
Cardiac hypertrophy	3
Hypoplastic circulatory apparatus	2
Hypoplastic external genitalia	5
Hypotrichosis	4

10. Of the 24 patients in the group, six were discharged from the hospital. Of these one was improved and five were unimproved.

11. Of the 24 patients, 12 died while in the hospital.

(a) *Causes of death:*

	Cases.
1. Status epilepticus.	
(a) Pneumonic complications	3
(b) Cardiac failure	1
2. Asphyxia	2
3. Post-convulsive cardiac dilatation	1
4. Lobar pneumonia	1
5. Pulmonary tuberculosis	2
6. Gastric hemorrhage	1
7. Septicemia	1

(b) Autopsies were performed on seven cases, revealing the following lesions of importance in the brain:

	Instances.
1. Cortical scars in brain	1
2. Cerebral arteriosclerosis	1
3. Lenticular softenings	1
4. No brain lesions	4

(c) Microscopical examinations showed the lesions described by Spielmeyer in four brains.

FEMALES.

1. Age on admission showed the usual variations.
2. Social condition showed the usual negro variations.
3. Formal education consistently below the eighth grade level.
4. *Heredity.*—

	Cases.
Grandparent, epileptic personality	1
Grandparent, epileptic	1
Parent, epileptic	1
Parent, eclamptic	1
Parent, alcoholic	3
Siblings, epileptic	1
Total	8

5. In two cases only was there a history of excessive alcoholism and this occurred previous to the onset of the convulsions. No history of other drug habits.

6. As a whole the economic, social and sexual adjustments were more disordered than in the foregoing groups, as in several cases the severe convulsions with changes in the personality began relatively early in life. The epileptic personality previous to onset of convulsions was present in only two cases.

7 *Convulsions.*—All patients were grand mal types with the convulsions beginning in infancy in nine cases; in three the fits began with puberty and menstruation. One patient had "hystero-epileptic attacks" in addition to the major convulsions.

8. *Outstanding Mental Symptoms.*—

(a) *Deterioration:* Present in all cases in its progressive form. No definite periods of confusion as such, other than that mentioned under "Metaphysical Delirium" were noted.

(b) *Metaphysical delirium:* Of the 28 patients four had a typical metaphysical delirium with the usual components.

(c) In only four cases were special fears and anxiety prominent with feelings of impending danger.

(d) *Sadism:* In only six of the 28 cases were sadistic activities in the foreground, which reaction is to be contrasted with its appearance in the group of confusion cases before described.

(e) Extreme *religiosity* was found in only one case, which is in keeping with the small number of cases in the males of this group.

(f) *Paranoid* projections were prominent in two cases.

(g) *Hallucinations:* Auditory hallucinations were noted in five patients, and no visual experiences were recorded excepting in those during the "metaphysical delirium."

(h) A serious depressive content was noted in three patients and one made an unsuccessful suicidal attempt by hanging.

(i) No catatonic states were noted.

(j) The remarks made before on superstitions in connection with convulsions and other episodes apply in this group.

9. *Physical and Laboratory Findings.—*

(a) All serology negative throughout the group, excepting two positive blood Wassermanns.

(b) *Neurosomatic deterioration syndromes* in various combinations were noted in seven cases.

(c) *Other physical findings:*

	Cases.
General syphilis	2
Closed and eroded sella turcica	5
Extreme anemia	1
Hyperthyroidism (Basedow's)	1

10. Of the 28 patients in the group, seven were discharged from the hospital. Of these four were improved and three were unimproved.

11. Of the 28 patients, nine died while in the hospital.

(a) *Causes of death:*

	Cases.
1. Status epilepticus.	
(a) Pneumonic complications	3
(b) Cardiac failure	1
2. Diabetic coma	1
3. Asphyxia	1
4. Pulmonary tuberculosis	1
5. Bronchopneumonia	1

(b) *Autopsies* were performed on five cases revealing the following lesions:

	Instance.
1. Hypoplastic circulatory system	1
2. Cerebral infarction	1
3. Diffuse brain gliosis	1
4. Polyendocrine sclerosis	1

(c) *Microscopical* examinations of two brains showed unusual number of rod cells, focal microgliosis in hyppocampus, "cicatricial infarcts" and hemorrhage into tissues, similar to those lesions found by Spielmeyer and by Minkowski in epilepsy.

C. Epilepsy with Mental Deficiency.

Of the 27 cases 15 were males and 12 females. We shall now consider the male group in detail.

MALES.

1. The majority of cases in this group were admitted relatively early in life.

2. The *social condition* showed the usual variations.

3. The majority of these cases were without school or other formal education.

4. *Heredity:*

	Cases.
Grandparent, epileptic	1
Parent, epileptic	2
Parent, psychotic	2
Parent, alcoholic	2
Siblings, epileptic	1
Siblings, tuberculous	1
Others, epileptic	2
Total	11

5. In three cases there was a history of excessive alcoholism previous to the onset of the fits. No drug habits were revealed.

6. All economic and social adjustments were inadequate. The "epileptic personality" was present in two patients, one of which was a murderer under sentence. One patient was a confirmed exhibitionist.

7. *Convulsions.*—All patients were subject to major attacks, and in ten cases they had appeared in early infancy. One convulsive reaction developed at puberty.

8. *Outstanding Mental Symptoms.*—

(a) No patient was psychometrically more than 10 years of age and several were low grade imbeciles.

(b) and (c) Of the 15 cases only two showed anything that might be interpreted as metaphysical delirium, and prolonged states of ambulatory confusion were not noted. In only one case were there any special fear reactions.

(d) *Sadism:* In 14 of the 15 cases sadistic activities were outstanding, and these patients as a rule were dangerous and difficult to nurse. Moreover, the sadistic behavior did not have the religious colorings noted in the confusion groups.

(e) Extreme religiosity dominating the personality was noted in only one case.

(f) Paranoid projections were noted in only one case.

(g) *Hallucinations:* Definite auditory hallucinations could be determined in only four cases and in three of these there were associated visual experiences.

(h) A serious depression was the notable feature in one case.

(i) A typical catatonic, negativistic reaction was noted in two cases.

(j) Very few superstitions or magical interpretations of various events were seen in this feebleminded group.

9. *Physical and Laboratory Findings.*—

(a) The serology was negative throughout, with the exception of one positive blood Wassermann.

(b) *Neurosomatic deterioration syndromes* were found in four patients. Of these two were parkinsonian expressions, and one was an ataxic cerebellar form of reaction.

(c) *Other physical findings:*

	Case.
General syphilis	1
Deformed dwarf	1
Cardiac hypertrophy	1
Cryptorchidism	1
Hypoplastic sexual organs	1
Infantile cerebral hemiplegia	1

10. Of the 15 patients in the group, five were discharged from the hospital. Of these two were improved in the number and severity of the convulsions and three were unimproved.

11. Of the 15 patients in the group, ten died while in the hospital.

(a) *Causes of death:*

	Cases.
1. Status epilepticus.	
(a) Pulmonary complications	2
(b) Cardiac failure	2
2. Typhoid with cerebral complications	1
3. Extreme emaciation	1
4. Acute dilatation of heart	1
5. Pulmonary tuberculosis	2
6. Acute hemorrhagic nephritis	1

(b) Autopsies were performed on four cases. The following lesions of importance were noted:

	Instance.
1. Internal hydrocephalus	1
2. Marked cardiac hypertrophy	1
3. Rickets	1

(c) Microscopically the lesions described by Spielmeyer and others were found in two brains.

FEMALES.

1. The majority of cases in this group were admitted early in life.

2. The *social condition* was variable, but most of the patients had never married.

3. These cases were largely without formal education or were not educable.

4. *Heredity.*—

	Cases.
Parent, epileptic	1
Both parents, epileptic	1
Parent, psychotic	1
Parent, syphilitic	1
Siblings, epileptic	1
Siblings, mentally defective	2
Total	7

5. No history of alcoholism or drug addiction.

6. All economic and social adjustments were inadequate. One patient was a confirmed pyromaniac.

7. All patients were subject to major attacks and in ten cases they had developed in early infancy.

8. *Outstanding Mental Symptoms.*—

(a) No patient was psychometrically over ten years of age and several were very low grade deficients.

(b) and (c) As would be expected there were no signs of confusion or metaphysical delirium as such. One patient had "running fits" and one had periodic fear states during which she was difficult to manage.

(d) In 7 of the 12 cases sadistic activities were in the foreground with similar reactions as in the male group of mental defectives. One five-year-old girl with a mental age of three years showed a peculiar destructive reaction. The patient had been irritable and restless at home. She beat herself so severely with her own fists that it was necessary to tie her hands. She tried in every way to injure herself, struck her head and any part of her body with clenched hands. Her face was alway badly bruised, with the eyes swollen. She always injured herself whenever her hands became untied in the night. (One wonders what these attacks mean in terms of self-destructive tendencies.) She had some sort of cry or scream which she occasionally uttered. If kept from beating herself, which was her chief occupation, she turned upon the one who was holding her hands, striking, kicking and attempting to bite. When she was held the impulse to strike was only restrained, for the muscles of her arms and legs were in constant play. If the restraining hand was moved a blow was delivered with the suddenness of a spring. She could not obey the simplest commands or requests.

(e) No religiosity was noted in this group.

(f) No paranoid reactions were noted.

(g) *Hallucinations:* Definite hallucinations were seen in only one case, and those were of the auditory type, being simple commanding voices of relatives.

(h) A serious depressive trend was noted in one case, which led to an attempt at suicide by jumping from a window.

(i) No catatonic reactions were noted.

(j) No superstitions or magical interpretations were particularly in evidence.

9. *Physical and Laboratory Findings.*—

(a) Serology was negative throughout the group. X-rays showed the sella turcica closed and eroded in two cases.

(b) *Neurosomatic deterioration syndromes* were found in five patients. Of these two were infantile hemiplegic anterior poliomyelitis combinations.

(c) *Other Physical Findings.*—

None at time of admission excepting one with strabismus.

10. None of these cases were ever discharged from the hospital.

11. Of the 12 patients in the group six died while in the hospital.

(a) *Causes of death:*

	Cases.
1. Status epilepticus.	
(a) Pulmonary complications	2
(b) Cardiac failure	1
2. Miliary tuberculosis	2
3. Acute pericarditis	1

(b) Autopsies were performed on four cases. The following lesions of importance were noted:

	Instance.
1. Congenital cerebral malformations	1
2. Microgyria	1
3. Small brain (850 gm.)	1

(c) Microscopical examinations of two brains revealed patchy changes in the cortical neurone cells, areas of arrested cortical lamination, minute cortical softenings, subependymal cysts, hippocampal gliosis, diffuse gliosis, and minute cortical hypoplasias of the cerebellum.

D. Epilepsy with Schizophrenic Components.

Of the seven cases, five were males and two females. The findings follow according to our usual procedure.

MALES.

1. Only one patient was admitted early in life (12 years); the others were in their adult years.

2. Three were married, two were single.

3. None had gone beyond the eighth grade in school.

4. *Heredity.*—

	Cases.
Parent, epileptic	1
Parent, cancerous	2

5. In two cases there was a history of excessive alcoholism previous to the onset of convulsions. No history of drug addiction.

6. The economic, social and sexual adjustments had been partly satisfactory in four cases.

7. *Convulsions.*—All patients were subject to major attacks, but in only one had they developed in early infancy.

8. *Outstanding Mental Symptoms.*—

(a) Schizophrenic components, such as active hallucinations, depersonalizations, odd, distorted behavior and catatonic tendencies were characteristic of the group.

(b) and (c) None noted.

(d) None noted.

(e) Nothing outstanding or excessive.

(f) Paranoid projections were present in two cases.

(g) *Hallucinations.*—Auditory hallucinations were present in all five cases and in two there were associated visual experiences. These hallucinations were chiefly concerned with voices of the dead, of the devil and of Jesus. Visions of angels and of hell were often reported.

(h) None noted.

(i) A typical prolonged catatonic reaction was noted in only one case, but temporary or short catatonic states were common in some of the others.

(j) None noted.

9. *Physical and Laboratory Findings.*—

(a) Serology negative throughout.

(b) *Neurosomatic deterioration syndromes*—none.

(c) *Other physical findings:*

	Case.
1. Cardiac hypertrophy	1
2. Hypoplastic external genitalia	1

10. Of the five patients in the group, four were discharged from the hospital. Of these three were improved and one unimproved.

11. No deaths.

FEMALES.

1. Both cases were admitted in early adult life.
2. Both patients were married.
3. Both had less than eighth grade education.

4. *Heredity.*—Negative.

5. No history of alcoholism or drug addiction.

6. In both cases the prepsychotic adjustment had been fair, although one had the "epileptic personality" make-up.

7. *Convulsions.*—Both had major convulsive attacks which began at puberty.

8. *Outstanding Mental Symptoms.*—

(a) Schizophrenic components were in the foreground as described for the males of this group.

(b) and (c) Both cases had a periodic metaphysical delirium reaction, but no interconvulsive fear states.

(d) *Sadism:* None noted.

(e) Extreme religiosity was noted in one case.

(f) No paranoid projections noted.

(g) *Hallucinations:* Both cases had definite and vivid auditory and visual hallucinations.

(h) No depressions noted.

(i) Catatonic states of moderate degree were seen in both cases.

(j) None noted.

9. *Physical and Laboratory Findings.*—All negative throughout.

10. One patient was discharged from the hospital as improved.

11. One died in status epilepticus with pulmonary complications. No autopsy.

E. Epilepsy with Organic Brain Diseases.

Of this small group of five cases, four were males, and one a female. We shall consider these cases without separation of the sexes.

1. All cases were in or past middle life when admitted.

2. All had been married.

3. None had been through eight grades at school.

4. *Heredity.*—

	Case.
Parent, insane	1

5. One patient had been excessively alcoholic before onset of convulsions.

6. Fair social and economic adjustment had been made by the four men, but the woman had been a poorly adjusted "epileptic personality" type.

7. *Convulsions.*—In one case the convulsions had started in infancy and in one at the age of 63, at which time the patient was suffering from some degree of senile dementia.

8. *Outstanding Mental Symptoms.*—

(a), (b) and (c) Mild confusions without metaphysical delerioid or fear components were noted in four of the cases. They were probably due to arteriosclerotic components.

(d) *Sadism:* This tendency was noted only in one case, the woman.

(e) Excessive religiosity was noted in one male.

(f) Paranoid projections were noted in one of the males.

(g) *Hallucinations:* Auditory hallucinations were experienced by two males.

(h) One man had depressions during one of which he expressed active suicidal ideas.

(i) and (j) No catatonic reaction or magic interpretations were encountered.

9. *Physical and Laboratory Findings.*—

(a) Serological examinations were negative throughout.

(b) *Neurosomatic deterioration syndromes* were present in two males.

(c) *Other physical findings:*

	Cases.
1. Generalized arteriosclerosis	3
2. Hemiplegia	1
3. Cardiac decompensation	1

10. No discharges.

11. All five cases died while in the hospital.

(a) *Causes of death:*

1. Status epilepticus.	Cases.
Cardiac failure	1
2. Cardiac failure	3
3. Cerebral hemorrhage	1

(b) Autopsies were performed on two cases, revealing the following lesions:

	Case.
1. Cerebral arteriosclerosis	1
2. Hemorrhagic pachymeningitis	1

IV. DISCUSSION AND CONCLUSIONS.

1. Age on admission varied greatly as it does in the white race.

2. The social condition showed the usual negro variations in marriages, illegitimacies and promiscuities.

3. The formal education had been less than the eighth grade throughout.

4. The usual difficulty in obtaining reliable family histories was encountered but in 85 of the 160 cases there had been combinations of epilepsy, major mental disorders, excessive alcoholism, and mental defect in the near relatives, usually in the parents or siblings. In 36 family histories there had been epilepsy in the grandparents or siblings.

5. In 36 patients there was a history of excessive alcoholism previous to the onset of the epilepsy or during its course or both. There was no history of other drug habits in any of the cases.

6. The economic, social and sexual prepsychotic adjustment varied greatly from none at all to fairly successful adaptation, however, actual misdemeanors and arrests were comparatively rare. The much mooted "epileptic personality" of Clark and others, with its grave, brutal, intense ill-humor, explosive violent passion, tendency to severe affect crises and adhesive, clumsy, bothersome, ceremonious behavior was alleged to have been present in the preconvulsive lives of 16 of the 160 patients (10 per cent).

7. *Convulsions.*—The age of onset, type of attack, frequency of convulsions, auræ, equivalents, etc., have been statistically considered in another paper; here it will suffice to say that all patients included in the study were subject to major convulsive attacks, the onset of which varied from early infancy to comparatively late in life. In 37 instances they had started in infancy and in 15 of the women the earliest seizures were associated with menstruation and the sexual epochs. There were two with so-called "running fits," one so-called "hysteroepileptic" and one with a migrainous-epileptic combination.

8. *Mental Symptoms.*—In addition to the 70 cases with outstanding confusion there were nine others affected to a milder degree.

	Cases.
Prominent progressive deterioration	52
Mentally deficient (less than 10)	27
Organic brain syndrome	7
Metaphysical delirium	33
Sadism and destructiveness	84

The sadism was seen principally in the confusion group and in the mental deficiency group. These acts against persons and things were frequently colored by religious ideas and sometimes motivated by such ideas. In the confusion the epileptic is freed from all traditional, moral and social discipline; he throws restraint to the winds and gives free expression to his sadistic impulses.

Extreme religiosity with mystical ideas concerning the activities and influence of God and the devil was prominent in 19 instances.

Paranoid projections ranging from mild complaints against those in the immediate environment to organized systems of persecutory delusions were found in 25 instances.

Olfactory and gustatory hallucinations were very rare, but auditory, visual and various somatic hallucinations principally although not wholly of religious content were found in 72 cases. Actual depressive content was seen in only nine cases, which is in keeping with the low rate of depressions among the negroes as a race. Suicidal gestures had appeared in five cases. No actual suicides occurred and moreover these five had a liberal admixture of white blood.

Magical and superstitious interpretations and odd, verbal expressions were encountered as outlined in the text; for instance, one patient thought when belching gas that he was giving birth to a monkey, another could feel men crawling into his flesh, and two others occasionally barked like dogs. There was considerable talk about voodoos and " strange spells."

9. *Physical Findings.*—

Syphilis.....4 instances (+ serology in blood only)

Apparently clinical syphilis was of little significance in the group.

	Instances.
Tuberculosis	10
Cardiac hypertrophy	8
Carcinoma	1
Eroded and closed sella	14

Hypoplasia of testes was not uncommon.

Neurosomatic deterioration syndromes (Hodskins) as evidence of disorder of the nervous system were demonstrated in 43 cases. There were various akinetohypertonic complexes with extreme muscular rigidity, progressive in the course of the epilepsy, and several combinations of pyramidal and extra-pyramidal phenomena with such symptoms as tremors of face, tongue and fingers, incoordination of muscles, diminished or exaggerated reflexes, increase in muscle tonus, unilateral paresis of muscles, contraction of flexor muscles of legs, contractions of ankles, ataxic gait, bilateral wrist drop, positive Romberg, positive Babinski, dulling of cutaneous sensations, tongue deviations, speech defects, aphonia, salivation and extreme emaciation.

There were two of the Parkinsonian life syndromes described by Hodskins and several with pseudobulbar types of disorder.

10. Forty-six of the 160 patients had been discharged from the hospital, 24 as improved and 22 as unimproved.

11. Seventy-seven of the 114 remaining patients have died in the hospital, 33 with status epilepticus, and the rest with asphyxiation, tuberculosis, pneumonia, cardiac failure, etc.

Autopsies were performed on 35 cases, the lesions ranging over a large number of somatic expressions. Perhaps the most important fact to mention here is the microscopical brain picture, described by Spielmeyer and stressed by Minkowski, consisting of a moderate diffuse cerebral neurogliosis with particular focal involvement of the hippocampus, and in addition cicatricial infarcts, minute hemorrhages into the tissues, increase in rod cells and neurone degenerations.

In a study of eight brains from cases of status or those dying soon after convulsions Minkowski found acute chromatologic changes in the purkinje cells with corresponding glial proliferations as described by Spielmeyer; but he failed to confirm the cellular alterations in Ammon's horn which Spielmeyer found in $\frac{1}{6}$ of the

cases dying in status. There were present, however, changes in the Betz cells, and fresh vascular changes such as dilated vessels filled with fresh clots, dilated perivascular spaces filled with amorphous tissue, round cells, and hemorrhages into the nervous tissue. The alterations in structure were diffuse, but showed a predilection for the subependyma, the cerebellar nuclei and the leptomeninges.

Since anatomic lesions are sometimes found in the brains of those who have had "genuine" or idiopathic" epilepsy for years and constitutional factors seem to play a rôle in symptomatic epilepsy, Minkowski believes that one should make a clinical distinction between these types only in the sense of the history in the individual case of actual factors that could produce epileptic seizures.

12. The study of the epilepsies in the negro race should indicate several avenues of approach to the subject in general, and in our next attempt we shall deal with those convulsive states which are partial expressions of organic brain disease.

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DISCUSSION.

DR. GEORGE VAN NESS DEARBORN (Bronx, New York).—I am quite sure that if Dr. Hubbard and Dr. Lewis get repeated psychometric examinations of undoubted epileptics, they will all show intellectual deterioration. In a number of years of experience with that sort of examination, I have never yet found a well-defined epileptic who did not show intellectual deterioration. I think it is part of the process, a mysterious part of a mysterious process.

I hope the series of papers which Dr. Lewis has in mind will lead to at least an elementary text book on negro psychiatry. I have for years been wondering, and asking some of my colleagues, why some of those who are familiar with the southern negro do not get together and write such a book relating to the negro race, and perhaps of a few other special races. But it is good to start with the negro, as he is in America.

I have been impressed frequently by the fact that in trying to make a diagnosis in the case of a negro suspected of psychosis or neurosis the things that interfered with an accurate diagnosis were their extraordinarily active fancy and imagination. I have repeatedly observed, as have others of my colleagues, that it may be almost impossible to tell whether a negro has hallucinations or not. Their imagination and fancy are so vivid that the dividing line between hallucinations and merely normal fancies is certainly very indefinite.

Paranoid projections, of which Dr. Lewis spoke, obviously involve the same difficulty and for just the same reason.

In these reasons alone there is sufficient sanction for the writing of an elementary text book by someone in the South or perhaps in Washington.

Although aware that Dr. Lewis has said that all of these cases of which he speaks were out-and-out epileptics, there is no doubt in my mind that there is some mode of continuity between "hysteria" and "epilepsy," as so often has been maintained. I should expect that there would be among negroes a relatively greater number of epileptic cases which are on the borderline, or just over the borderline in hysteria, owing to the hypersuggestability of the negro race.

I have, like you, been in the habit of thinking of them as averaging about ten points lower than the other races in their intelligence quotients, and their diagnoses should be adopted in sundry ways to this difference.

DR. LEWIS.—A book could be written on the basis of the material we have. What I hope to do eventually is to write 15 or 20 papers in detail and then see if they can be arranged into book form.

The negro is different from the standpoint of nurture and culture and his reactions at these levels should be carefully studied. I think that Garth and others who have done a great deal of psychometric testing feel that there are no very great fundamental differences in any of the races so far as those measurements are concerned. What I fear is that they are not measuring the important things. There are certain personality traits that they are not as yet able to get into a metric approach, and they are often the most important

ones. There are certainly differences from the standpoint of the surface expression of emotions, and perhaps also from the standpoint of the fundamental meanings.

I agree with Dr. Dearborn that all epileptic subjects do show deterioration. By this particular way of grouping, I meant to indicate in one group, those cases showing a gradual progressive deterioration without those other striking features that are frankly psychotic and emotional, or the periodic activities and equivalents which in themselves lead to the institution.

Regarding hystero-epilepsy, I have not particularly studied the possibilities implied in that diagnosis; but four or five years ago no less an authority than Professor Bleuler went into the subject very carefully—all of the work that had been done and all of the literature—and in one of his annual addresses in Paris, he stated his conclusion that there is no such thing as hystero-epilepsy, that it is a misnomer, that a case is either hysteria or epilepsy and that we have no more reason to say "hystero-epilepsy" than to use the term "typho-tuberculosis" for a case with these diseases combined. That is Professor Bleuler's attitude, which I am inclined to accept.

DR. GEORGE VAN NESS DEARBORN (Bronx, New York).—I cannot see what Dr. Lewis means by saying the case is either epilepsy or hysteria. We have patients in staff conferences frequently, in whose cases an intelligent group of medical men are absolutely unable to decide whether to diagnose hysteria or epilepsy.

I, for one, would like to inquire the criteria on which Dr. Lewis differentiates in such a perfect manner between hysteria and epilepsy, as we know hysteria nowadays.

DR. LEWIS.—Personally I have not had an extensive experience with cases of so-called hystero-epilepsy; that is, hysteria with convulsions or epilepsy with hysteria forms. Although we have a very large service and clinic at St. Elizabeths Hospital, such cases are very seldom encountered.

I do not think that there is any sort of mental or neurological condition that may not at some time have hysteria or hysteroid reactions. I believe that the attitude of Prof. Bleuler should be carefully considered by anyone dealing with such cases.

CLINICAL AND EXPERIMENTAL OBSERVATIONS ON THE BABINSKI REVERSAL, CARDIOVASCULAR REACTIONS, RESPIRATORY AND PUPILLARY CHANGES DURING THE CONVULSIVE AND POST-CONVULSIVE STAGES OF GENERAL AND EXPERIMENTAL EPILEPSY.*

By J. NOTKIN, M. D., HELEN C. COOMBS, Ph. D., and F. H. PIKE, Ph. D.

CONTENTS.

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* Read at the eighty-seventh annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Toronto, Canada, June 1, 1931.

The clinical observations recorded in this paper were made in the wards of the Manhattan State Hospital, and the experiments on animals were done in the laboratory of physiology of the New York Homeopathic Medical College. Some control experiments, to which reference is made, were done in the neuro-surgical laboratory of Columbia University. The expenses of these were defrayed by a grant from the Commonwealth Fund to the Neurological Institute of New York.

GENERAL STATEMENT

The object of this paper is to present the results of a study of the kind and order of events in the process known as an epileptic seizure, and their comparison with the conditions encountered in convulsions of experimental origin in animals (cats).

It is highly desirable to get as complete and accurate an account of these events as possible if any analysis or explanation of the process of the convulsion itself is to be made. We are avoiding, so far as we can, the use of the term "idiopathic," as signifying that the disease is its own cause or its own pathology, since this is an archaic term and not in accord with the facts. In the present paper we shall consider the clinical findings in the period following the last clonic twitch, and the experimental observations during the whole period of the convulsion.

There are a number of phenomena to be observed in the convulsive states immediately after the last clonic movement and while the patient is still unconscious. Some of these are practically always present, while others appear only occasionally. To the first group belong the Babinski sign, changes in pulse rate and blood pressure, and sometimes pupillary changes; to the second, and coming somewhat later, sleep states, confusional episodes and transitory sensory and motor manifestations such as muscle sense changes, agnosia, temporary decrease of auditory acuity, even to the point of deafness at times, concentric limitations of the visual fields, particularly for red and green, hypotonia, transitory loss of deep reflexes, and, occasionally, monoplegias of short duration. Sleep may or may not occur in non-psychotic epileptics and in epileptics with mental complications. Confusional states, however, occur chiefly in psychotic patients. This state may last from a few minutes to several hours or even days. We shall not discuss in detail at present the post-convulsive changes which are not constant, but shall limit ourselves chiefly to the consideration of the reactions which are practically always seen, and which follow, as a usual thing, immediately upon the clonic phase of the convulsion.

The clinical material for this study was drawn from the wards of the Manhattan State Hospital, and consisted of 69 patients without obvious organic lesions of the central nervous system or obvious pathology of the cardiovascular system. The animals used in the experimental procedures were cats. The technique of the various

types of experiments has been described so frequently in previous papers as to require no further comment here.

RESULTS: CLINICAL AND EXPERIMENTAL.

The Babinski Phenomenon.

The occurrence of the Babinski sign after an epileptic convulsion is well known to every clinician. It is frequently mentioned in clinical reports, but there are relatively few investigators who have studied the frequency or constancy of its occurrence, its uni- or bilaterality, its duration and the exact time of its appearance. Some of the data in the literature on this subject are rather confusing, as not infrequently an author speaks of a dorsal extension of the whole foot or of all the toes; others do not specify the time of the appearance of the sign except to state that it was observed "during a convulsion"; while a few investigators failed to observe the Babinski sign after a seizure. After a brief review of the literature in chronological order, we shall give our own observations.

Babinski (1898) was the first to elicit, in a case of Jacksonian epilepsy, the dorsal extension of the toe on the involved side immediately after the convulsion. In the following year (1899), he drew attention to this dorsal extension of the toe during the attacks in cases of so-called "idiopathic" or general epilepsy. Babinski did not specify, however, any special phase of the convulsion during which the dorsal extension of the great toe could be elicited.

Collier (1899) observed the Babinski sign immediately after an attack in 38 out of 40 cases, that is, in 95 per cent. The sign could be elicited for a period of four or five minutes. In one case with 40 attacks at brief intervals, he observed dorsal flexion of the toes seven hours after the last attack.

Cestan and LeSourd (1899) saw the Babinski sign during an attack in two out of thirteen cases (15 per cent), and Koenig (1900) observed it once during the state of epileptic coma. De Pastrovich (1900) found a bilateral Babinski sign after the attacks in a 35-year-old woman who had five or more severe convulsions daily. Crouzon (1900) elicited the Babinski sign in 41 per cent of his cases, but he does not specify the phase. In another group of 91 cases, he observed the dorsal extension of the great toe in 16 cases half an hour or more after the convulsion.

Homburger (1901) and Schneider (1901) failed to elicit the Babinski sign during or after a convulsion. Esmenard (1902) reports instances where there was at first no response to plantar stimulation during an attack, but later a flexor type of response. In other cases, he observed no response at first, and then a dorsal extension of all the toes. In another group of cases, he reported instances of a dorsal extension of all the toes during the whole period of the convulsion.

Specht (1903) observed the Babinski sign eight times after convulsions in a case of symptomatic epilepsy (cerebral arteriosclerosis); the sign would last from one to one and a half minutes. Kenniston (1903) found extension of the foot bilaterally in 95 per cent of his subjects during attacks, and in 18 per cent one hour later.

Redlich (1906) believes that the Babinski sign can be elicited uni- or bilaterally, after a single attack, for minutes and even hours, and, after repeated convulsions, for days.

Finkelnburg (1908) observed in 19 out of 23 cases (19 per cent) uni- or bilateral Babinski responses lasting from a few minutes to 15 minutes after an attack. In one case, he elicited the Babinski sign three hours after the attack. In the cases with petit mal attacks, he saw a positive Babinski sign in only one out of four instances. Finally, Hempel and Berg (1910) found the Babinski sign in 86 per cent of their cases, more often bilaterally.

Personal Observations.

In order to obtain more detailed data, we have studied the Babinski phenomenon in a group of epileptic patients at the Manhattan State Hospital for a period of several years. Although our observations were numerous, we had to discard more than 100 records as we were not always able to be present during the whole period of the convulsion, and thus were unable to record accurately the time of the initial appearance of the Babinski sign. In order to avoid mistakes and faulty interpretations, we have used for this communication observations on 69 different patients which we studied carefully during the whole phase of the convulsion and for some time immediately afterward. Moreover, we have limited

ourselves to cases of the so-called "cryptogenic" variety of the convulsive states; in none of them were the Babinski or other neurological signs present in the intervals between the convulsions. Before proceeding to the consideration of our own findings, we would like to emphasize not only the theoretical importance of the Babinski sign, but also its great practical value in differentiating seizures of the petit mal type without gross manifestations from various attacks of a cardiovascular nature like fainting spells, dizziness, syncopies, and the like. L'Hermitte and Dupont (1929) however, observed in a patient of 63 years, suffering from intermittent attacks of cardiac insufficiency, the appearance of the Babinski sign during the attacks. The pathology of this case is uncertain. The sign is also of importance in the differentiation of an epileptic attack from the seizures seen in cases of pure hysteria, although in the latter group a gradual transformation into epileptiform organic convulsions with a positive Babinski sign is quite possible. We have referred to this in the study of "Affectepilepsy and Hysteroepilepsy" (Notkin, 1930).

It is important to note here that the earliest period at which we were able to elicit the Babinski sign was at the beginning of the stage of relaxation, immediately after the cessation of the clonic movements. We did not see the Babinski sign during the tonic phase of the convulsion; in the clonic phase, the movements of the limbs are sometimes so violent that it is hardly possible to make an accurate estimation. We are inclined, however, to believe that there is no Babinski sign during the clonic phase, particularly if we bear in mind the fact, as we shall note later, that in 43.1 per cent this sign was somewhat delayed and did not follow immediately after the cessation of the convulsion. In fact we have observations in which even a flexor response to plantar stimulation was noted in the first two minutes during the phase of relaxation.

It would take up too much space to reproduce here all our observations; we shall give therefore only a few records illustrating various types of reactions.

Of the 65 observations 64 gave a positive Babinski sign and in only four instances, or 5.8 per cent, did we fail to elicit the Babinski sign upon plantar stimulation after the cessation of the clonic phase. We had opportunity to observe the same patients during a number

of convulsions with similar results. The following record gives the observations in a typical case:

MARGARET McC.

3.15 P. M.—Generalized clonic convulsion; no plantar response to stimulation.
3.16 " —Stage of relaxation; no plantar response.
3.17 " —No plantar response on either side.
3.19 " —Lethargic; flexor response to plantar stimulation on both sides.
3.20 " —*Idem.*
3.21 " —*Idem.*
3.23 " —Regaining of consciousness; strong flexor response.

The Babinski sign was not elicited in all instances immediately after the last clonic twitch, or after the cessation of the tonic phase in the cases where the tonic element alone was present. In 28 out of the 65 cases (43.1 per cent), there was a lapse of from one to three minutes during which time there was no response of any kind to the plantar stimulation; and in a few instances, we observed a flexor response in the first two minutes, followed by a strong Babinski sign in the succeeding minutes. The following record may serve as an illustration of this type of reaction:

MARY L.

10.48 A. M.—Generalized convulsion; no plantar response to stimulation.
10.49 " —Relaxation; no response.
10.50 " —Bilateral flexor response.
10.51 " —Strong bilateral Babinski.
10.52 " —Strong bilateral Babinski.
10.53 " —Regaining consciousness; flexor response on both sides.

In the majority of instances in this group of 28 observations, there was at first no response whatever for two minutes; the Babinski sign appeared afterwards, as is seen in the following record:

ELIZABETH B.

10.01 P. M.—Generalized convulsion; no plantar response to stimulation.
10.02 " —Relaxation; no plantar response on either side.
10.03 " —*Idem.*
10.04 " —Weak Babinski on both sides.
10.05 " —Strong Babinski sign on the left side, slightly weaker on the right side.
10.06 " —Strong bilateral Babinski.
10.07 " —*Idem.*
10.08 " —*Idem.*
10.09 " —Babinski sign weaker on both sides.
10.10 " —Regaining consciousness, flexor response on both sides.

As has been mentioned, in a little more than half of the cases, that is in 37 observations or in 56.9 per cent, the Babinski sign appeared upon plantar stimulation immediately after the last clonic movements:

KATE K.

9.36 A. M.—Last clonic twitches, no plantar response.
9.37 " —Relaxation, bilateral Babinski on plantar stimulation.
9.38 " —*Idem.*
9.39 " —Left Babinski stronger than right.
9.40 " —Babinski on both sides weaker.
9.41 " —Regaining of consciousness, flexion of big toes on both sides on stimulation.

We were not able to elicit a bilateral Babinski sign in all instances. In 17 cases, or 26.1 per cent, the Babinski sign could be elicited on one side only. In the majority, however, 48 cases, or 73.9 per cent, the Babinski sign was present on both sides. The following is an example of the unilateral response:

FRIEDA H.

10.25 A. M.—Relaxation immediately after cessation of the convulsion; no plantar response.
10.26 " —Babinski on the left side, flexion on the right side on plantar stimulation.
10.27 " —Strong Babinski on the left side, flexion of the right.
10.28 " —*Idem.*
10.29 " —Weak Babinski on the left, flexion on the right.
10.30 " —Regaining consciousness; flexion on both sides.

In several cases with a positive Babinski response, we examined also for other signs, such as absence of the abdominals, the Chaddock and the Hoffmann signs; in all observations where the Babinski was positive, the other signs were also positive. We give below one illustration with the Hoffmann sign:

MABEL R.

1.55 P. M.—Clonic convulsion; no plantar response.
1.55½ " —Relaxation; no plantar response.
1.56 " —No response.
1.57 " —Strong right Babinski and weak left Babinski.

TABLE I.

SHOWING THE AGE OF ONSET, THE AGE AT WHICH THE EXAMINATION WAS MADE, THE DURATION OF THE DISEASE AND THE DURATION OF THE BABINSKI REVERSAL IN A SERIES OF CASES.

Age of onset.	Age at examination.	Duration of the disease.	Duration of Babinski sign.	Age of onset.	Age at examination.	Duration of the disease.	Duration of Babinski sign.
		Yrs.	Min.			Yrs.	Min.
?	38	?	1	15 yrs.	40	25	1
17 yrs.	40	23	3	Inf.	31	30	6
13	39	26	0	19	26	7	3
16	43	27	2½	?	24	?	0
20	33	13	3	Inf.	32	31	10
6 mos.	37	36	5½	23	28	5	5
7	23	16	2	18	35	17	10*
Inf.	36	35	7	?	41	?	24
12	20	8	4	Inf.	39	38	2
16	40	24	1*	9 mos.	24	23	1
11	36	25	3	15	26	11	4
20	30	10	6	11	23	12	½
5 mos.	31	30	1	19	38	19	10
Inf.	33	32	5	10	25	15	5*
?	36	?	½	19	32	13	11
16	24	8	3	8 mos.	26	25	3
14	25	11	1½	Inf.	39	38	1
?	35	?	2½	15	26	11	2
?	37	?	1	20	29	9	8
?	41	?	6	5	32	27	2½
Inf.	28	27	5*	Inf.	27	26	20
4	35	31	2½	15	32	17	1½*
7	37	30	1½	13	36	23	12
?	33	?	0	Inf.	43	42	3
19	26	5	3	16	35	19	6
17	32	15	1	9 mos.	41	42	½
?	34	?	10	17	29	12	1
?	37	?	0	12	33	21	0*
Inf.	34	33	½	14	35	21	4
?	29	?	7	16	29	13	5
15	34	19	1	7 mos.	32	31	3
7	31	25	2	Inf.	28	27	8
?	29	?	3	15	35	20	7
17	33	16	8*	9	41	32	17
12	36	24	4				

* Indicates that the patient has died since the date of examination.

1.57½ P. M.—Strong Babinski on both sides and strong Hoffmann both sides.
1.58 " —*Idem.*
1.59 " —Strong right Babinski, flexion on left, Hoffmann both sides.
2.00 " —*Idem.*
2.01 " —Regaining consciousness, flexor plantar response, Hoffmann absent on both sides.

The duration of the Babinski sign, as seen from Table I, varies, in our cases, from one-half minute to 25 minutes.

The number of cases for each particular duration observed in this series is given in Table II. The table indicates a bimodal dis-

TABLE II.

DISTRIBUTION OF CASES SHOWING THE BABINSKI SIGN ACCORDING TO DURATION.

Number of cases.	Duration of the sign, minutes.
4	½
10	1
3	1½
4	2
4	2½
10	3
4	4
5	5
1	5½
4	6
3	7
3	8
4	10
1	11
1	12
1	17
1	20
1	24
1	25

tribution, with 10 cases showing a duration of one minute, and again 10 cases showing a duration of three minutes.

The significance of the varying duration of the Babinski sign is unknown, but from the known facts of the progress of the disease, one might expect to find the greatest duration in the most severe cases or in those cases which have persisted for the greatest length of time. In Fig. 1, we have plotted the duration of the Babinski reversal of the plantar reflex against the age of the patients at the date of examination. The age is given in the vertical column at

the left, and the duration is given along the horizontal line at the bottom of the figure. Except for two aberrant cases, one at age 20 and one at age 27, the distribution of the circles indicating the duration of the Babinski sign for any given age shows an increasing duration with the increasing age of the patient. It is possible that the expectation of life in those patients in which the duration of the Babinski sign is indicated by the circles lying farthest to the right is significantly less than in the case of the other patients in which the duration of the sign is indicated by the more closely grouped circles.

The Babinski sign disappears in three minutes or less in 54 per cent of the cases. In only 15 per cent did it persist 10 minutes or longer. In 29 per cent the duration was from four to eight minutes.

It is of interest to note that the Babinski sign is at first weak, that it becomes gradually stronger, and then weak again before its final disappearance, which occurs always at or just before the moment of regaining consciousness. At no time were we able to elicit the Babinski sign after the return of consciousness, which is contrary to the findings of some other observers.

Any attempt to explain the mechanism of the Babinski reversal in our own or other observations will necessarily remain futile, since all theories regarding this reversal are still far within the realm of speculation. In recent years there has been a tendency to consider the possibility of a revival of paleogenetic function when the phylogenetically newer structures of the central nervous system are no longer intact. This opinion was also expressed by Steiner (1885) and by Pike (1909) in his studies on the general phenomena of spinal shock. Von Monakow (1914) utilized the same theory in an attempt to explain the mechanism of the Babinski reversal without, however, giving any definite indication toward what system the "shifting," as he calls it, of this particular function is taking place. Rabiner and Keschner (1926) made an attempt to explain the mechanism of the Babinski sign as a release of extra-pyramidal manifestations. They develop their theory on the basis of the development of the pyramidal system in higher vertebrates, and particularly in man, at the expense of the extra-pyramidal. They say ". . . . the dorsiflexion of the big toe is the posture in animals whose extra-pyramidal system is the predominant efferent pathway . . . . with the development of the pyramidal system, the foot

becomes plantigrade with the big toe as the fulcrum for the erect posture." In the intact central nervous system, both the pyramidal and the extra-pyramidal systems are in balance and the big toe is neither in flexion nor extension. When, however, the influences of the pyramidal system are eliminated, the extra-pyramidal influence comes into play with a reversion to the old posture. They argue further, "Although dorsiflexion of the big toe is indicative of disturbance of the pyramidal influences, it is also evidence of a preponderance of extra-pyramidal influences."

We may remark in passing that there are grave objections to regarding the pyramidal and the extra-pyramidal systems as antagonistic, or even as functionally separate systems. (Pike, *et al.*, 1931). And the so-called " release " phenomena present an insoluble puzzle to the physiologist unless one thinks of them in terms of changes in the quantities of nervous energy passing over given conduction systems.

Another attempt to explain the pyramidal tract signs was made by Wechsler (1923), who observed several cases of the myoclonic form of encephalitis associated with loss of the abdominal reflexes and with psychotic symptoms; on the basis of his observations, he advanced the possibility of the passing of the central arc between the afferent and efferent (pyramidal) systems through the frontal lobes, suggesting that the latter structures are the seat of all psychic functions. He explains the loss of the abdominal reflexes and the mental manifestations by the involvement of the frontal lobes. Attractive as this theory may be, it can hardly be applied to our cases, since the Babinski sign was elicited only during the stage of unconsciousness, and not when the patients were merely confused. It is probable that some other pathway than the one through the frontal lobes was blocked. It is of interest to note that at no time were we able to elicit a dorsal extension of the toe in epileptics or other patients who were soundly asleep, or in patients during the stage of surgical narcosis. Kleitman (1923), however, reports that in the severe exhaustion following prolonged loss of sleep, the Babinski reversal of the plantar reflex may occur.

Experimentally the Babinski reversal in the hind foot of a cat has been observed on the side of the lesion in a convulsion elicited by the intravenous injection of absinth after experimental hemisection of the spinal cord. (Pike, 1928.)

The most one can say for the time being to explain the occurrence of the Babinski sign in the post-convulsive period is to suggest the possibility of some derangement, more or less temporary, of the pyramidal system due to severe stimulation during the convulsive attacks, perhaps something similar to a pyramidal tract involvement of a fleeting character.

The problem of the Babinski reversal of the plantar reflex is one phase of the problem of the functional organization of the motor system in general; and its irruption into the field of the problem of the convulsive states is merely another indication of the truth of the view that the problem of the motor phenomena of the convulsive states is merely one phase of the wider problem of the functional organization of the motor system in general. Our knowledge of both is still incomplete, and our views still highly tentative, where they are not merely speculative, because of the incomplete functional analysis of the central nervous system itself.

Cardio-Vascular Changes.

clinical observations.

Other phenomena of interest during the convulsive or post-convulsive periods are the pulse rate and blood pressure variations. With reference to the pulse rate, we found next to nothing in the literature presented in a form which could be used. More attention has been paid to the blood pressure changes, although here again we find a lack of precise statements. Nor have we found any presentation of both blood pressure and pulse rate which would enable one to determine the correlation between the two factors during or after a convulsion. Ohannessian and Lugiato (1906) reported their observations on the blood pressure changes during convulsive seizures, stating that the blood pressure rises from 170 to 230 millimeters of mercury, with an average of 200 mm. In the following year (1907) Ohannessian again stated that the blood pressure rises considerably during the clonic phase, only to fall below normal at the moment when the convulsion ceases. Lallement and Rodier (1909) reported a rise of pressure during the convulsion up to 210 or 220 mm. of mercury. They also observed an increase in the pressure shortly before the onset of a convulsion.

It has been our experience that, during a convulsion, in the tonic and clonic phases alike, the pulse becomes practically imperceptible and a determination of the rate is impossible. Immediately after the cessation of the clonic phase, however, the pulse rate is up strikingly, and sometimes attains a rate twice that of the interparoxysmal period.

Space does not permit giving the records of more than a few observations.

MARGARET C.

(Interparoxysmal pulse rate 86.)

	Time.	Pulse rate.
Clonic phase	2.35	Imperceptible
Clonic phase	2.36	Imperceptible
Clonic phase	2.37	Imperceptible
Relaxation	2.38	140
	2.39	145
	2.40	135
	2.42	130
	2.44	130
	2.46	128
	2.48	100
	2.49	98
	2.50	98
	2.53	94

Nor were we able to make any blood pressure readings during the tonic and clonic phases. Upon cessation of the clonic movements we have observed an increase of the systolic pressure in slightly above 50 per cent of our cases; in the rest, there was a considerable drop. The diastolic pressure remains normal in the majority of cases, with an increase in the systolic pressure. In the instances in which there was a lowering of the systolic pressure, the diastolic always showed a marked fall, sometimes to zero. In the latter group of cases, there is a recovery of both the systolic and diastolic pressure in the fourth to the tenth minute after cessation of the clonic movements. The pulse seems to regain its approximately normal rate coincidently with the recovery of blood pressure, while the Babinski sign continues positive, as is shown in the last of the three following records.

ANNA McC.

(Interparoxysmal blood pressure 110/76; pulse, 86.)

	Time.	Pulse rate.	Blood pressure.	Babinski sign.
Cyanosis and frothing.	2.35	Imperceptible	?	Negative.
Relaxation	2.36	135		Positive, bilateral.
	2.37		190/60	
	2.38	130		
	2.42		180/60	
	2.44		160/70	
	2.45	128		
	2.46		130/80	Positive, bilateral.
Regaining consciousness	2.47	96	130/80	Negative.

AMELIA H.

(Interparoxysmal blood pressure, 120/80.)

	Time.	Blood pressure.
Stage of relaxation.....	1.43	160/0
	1.44	160/0
	1.45	140/0
	1.46	138/0
	1.48	110/40
	1.55	125/40

ANNA H.

(Interparoxysmal blood pressure 118/70; pulse 84.)

	Time.	Pulse rate.	Blood pressure.	Babinski sign.
End of clonic phase...	4.41	Imperceptible	?	Negative.
Relaxation	4.42	110		
	4.45			Positive, bilateral.
	4.46		95/0	
	4.47			Positive, bilateral.
	4.50		100/0	
	4.52			Positive, bilateral.
	4.53	102		
	4.55			Positive, bilateral.
	4.58		100/0	
	5.01		110/60	
	5.02	102		
	5.03			Positive, bilateral.
	5.09		110/60	
Regaining consciousness	5.11	96		Negative.

As we cannot give here in detail all our records of pulse rate and blood pressure, we give a table (Table III) of the findings in a group of 30 patients during the interparoxysmal period, during the phase of relaxation and at the return of consciousness.

As previously stated, there was a rise of blood pressure during the phase of relaxation in slightly more than 50 per cent of the cases, and a fall in the others. We do not offer any explanation of this difference; the examination of the cardio-vascular system gives no clue, as in no case was any evidence of cardio-vascular pathology discovered. The pulse rate, on the other hand, always rises during the phase of relaxation, being higher than during the interparoxysmal period.

The interparoxysmal blood pressure readings received considerable attention in an attempt to ascertain whether there were any variations prior to a convulsion. Such studies were made by Marchand and Adam, Hartenberg, Reed, Trentzsch, and others. The findings are contradictory, and we are much in doubt as to whether there are any records which were made shortly before a convulsion. We say this on account of our own disappointing experience. We sat up for hours, and on one occasion for a whole day, with a patient, taking blood pressure and pulse rates at short intervals, and we never succeeded in obtaining such records at any time close to a convulsion. The findings in a particular group of patients whose blood pressure was taken at intervals of 10 or 15 minutes, are not characteristic. Except for slight changes, there were no variations in the blood pressure or pulse rate in a period of several hours.

The comparison between the cardio-vascular conditions in the phase of relaxation following a convulsion, as given in Table III, and conditions in the same patients during the interparoxysmal period may be shown strikingly by graphic means. If the pulse rate is plotted vertically and the blood pressure (systolic) horizontally, as in Fig. 2, the cardio-vascular condition of any given patient may be represented by a point located at the intersection of the vertical line through the point on the horizontal axis representing the blood pressure of that particular patient, and the horizontal line through the point on the vertical axis representing the pulse rate of that patient. Thus, if the blood pressure and pulse rate of a given patient during the interparoxysmal period are 110 and 80,

TABLE III.

SHOWING THE AGE OF ONSET, THE AGE AT EXAMINATION, THE DURATION OF THE DISEASE, THE PULSE RATE AND BLOOD PRESSURE DURING THE INTERPAROXYSMAL PERIOD AND DURING THE PHASE OF RELAXATION AFTER A CONVULSION.

No.	Age at onset.	Age at examination.	Duration of disease.	Blood pressure during.			Pulse rate during.		
				Interparoxysmal period.	Phase of relaxation.	Return of consciousness.	Interparoxysmal period.	Phase of relaxation.	Return of consciousness.
1	19	32	13	110/76	190/60	130/80	86	135	96
2	8	36	28	120/80	160/0	125/40	...	...	...
3	4	35	31	130/80	165/70	135/75	90	145	94
4	Inf.	39	38	126/78	170/50	134/56	80	135	86
5	15	35	20	118/76	160/60	128/65	76	140	82
6	16	29	13	135/84	180/50	140/50	86	150	92 *
7	14	35	21	120/80	145/40	130/60	72	130	86
8	20	29	9	110/80	180/70	125/50	80	125	90
9	3	38	35	118/70	145/60	130/0	86	130	92
10	17	40	23	120/76	160/50	125/65	90	140	100
11	13	39	26	124/80	190/50	140/0	...	...	...
12	14	25	11	116/66	180/45	130/70	76	145	88
13	20	36	16	124/80	160/70	135/65	84	132	90
14	16	40	24	96/60	135/40	100/0	60	100	80
15	Inf.	33	32	118/72	180/60	130/60	86	135	90
16	12	26	14	120/82	170/66	134/60	82	120	89
17	16	33	17	118/74	100/0	110/60	90	102	96
18	?	33	?	118/70	95/0	110/60	84	110	96
19	15	40	25	120/80	90/0	100/40	86	120	100
20	19	26	7	110/66	85/0	110/50	72	100	86
21	18	35	17	100/60	94/0	100/40	80	96	86
22	Inf.	40	39	126/84	100/0	110/40	...	...	...
23	7 mos.	37	30	112/70	88/0	98/40	86	110	92
24	6 mos.	37	36	118/86	98/0	100/60	90	120	106
25	16	40	24	110/72	98/0	106/60	82	130	94
26	?	34	?	120/70	86/0	110/70	...	...	...
27	14	35	21	116/74	90/0	108/40	96	130	100
28	8 mos.	26	25	90/0	80/	90/	90	120	94
29	15	35	20	110/80	96/0	...	84	110	98
30	7	31	24	108/68	90/0	100/50	88	134	96

* Patient has died since examination.

respectively, then that patient may be represented by a small circle at the intersection of the horizontal line through 80 at the left of the chart with the vertical line through 110 at the bottom of the chart. All of the blood pressures and heart rates taken during the interparoxysmal period are thus represented by circles in Fig. 2. Where two patients have had the same blood pressures and heart rates, the circles have been superposed. Similarly, the data on blood pressure and pulse rates taken during the phase of relaxation are represented by squares. In those cases in which a rise of blood pressure occurred during the convulsion, as indicated by the readings during the phase of relaxation, both the circles and the squares have been filled in solidly black. Conversely, in those cases in which the blood pressure fell during the convulsion, the circles and squares have been left open. The filled and open circles, representing the conditions during the interparoxysmal period, are well grouped together, but there is a clear and unequivocal separation into two groups during the phase of relaxation.

It is of interest to note that the patient (Anna H., number 18 in Table III) in whom the Babinski reversal persisted longest—25 minutes—comes in the group in which the blood pressure fell during the phase of relaxation.

Inasmuch as blood pressure, pulse rate and duration of the Babinski sign were not all taken on all patients, we cannot draw any general correlation between the findings in the two groups of patients in Table I and Table III. In another case (Anna McC., number 1 in Table III), the duration of the Babinski sign was 11 minutes; the blood pressure increased from 110/76 to 190/60 during the convulsion, and the heart rate rose from 86 to 135.

On purely statistical grounds, one might expect intermediate types which would fill in a Y-shaped distribution of the squares indicating pulse rate and blood pressure during the phase of relaxation. It is probable that, with a greater number of cases, such a Y-shaped distribution might be obtained. If we may anticipate here some results to be presented in later sections of this paper, we might even get a complete wreath of squares surrounding the group of circles which represent the conditions during the interparoxysmal period.

EXPERIMENTAL OBSERVATIONS.

The detailed findings on the cardio-vascular changes in convulsions of experimental origin are presented in another place (Coombs and Pike, 1931). We shall give here only some of the more important facts which bear upon the question of the interpretation of the clinical observations. For the sake of clearness, we consider the blood pressure changes separately.

The animals (cats) which were used for experiment were etherized when operations of any considerable magnitude, *e. g.*, transection of the spinal cord, were done before taking blood pressure. In all cases, except those of one or two control animals, the ether was intermitted an hour or more before the injection of camphor monobromide or absinth. The technique of this injection has been given elsewhere (Pike, Elsberg, McCulloch and Rizzolo, 1929) and need not be repeated here. Blood pressure was usually taken from the femoral artery by means of a cannula in the artery connected to a mercury manometer. In one experiment, blood pressure was taken from one carotid artery.

Respiratory movements were recorded through a stethograph attached at the level of the diaphragm and connected to a tambour whose writing point was in contact with the kymograph paper directly above the writing point of the blood pressure manometer. Curare, adrenalin and ephedrine, when used, were injected directly into the femoral vein of one side.

The experiments may be grouped as follows:

1. Experiments in which the animal was anæsthetized at the time of injection of the convulsant agent.

The blood pressure falls in all such cases, and there is no convulsion. (Fig. 3.) Absinth and camphor are listed in the literature of pharmacology (*e. g.*, Sollman, 1917) as vasodilator agents, and a fall of mean arterial pressure is to be expected under these conditions. The fall is merely transitory, however, and the pressure soon returns to normal or control level.

2. Experiments on curarized animals without general anæsthesia.

In all these experiments, the general arterial pressure fell on injection of absinth or camphor monobromide. Even a moderate dose was sufficient to bring about a fall of blood pressure to a level which was insufficient to maintain the action of the heart, and death

resulted. The intravenous injection of adrenalin during the period of low blood pressure following the administration of the convulsant agent induced only a transitory rise of blood pressure and was ineffectual in restoring the animal to a condition in which the heart would continue to function effectively. The vasomotor mechanism appeared to be severely damaged by the absinth or camphor monobromide.

3. Experiments in which the spinal cord was transected before the injection of absinth or camphor monobromide.

The effects vary somewhat according to the level of the transection.

When the transection was made as high as the level of the second or third thoracic segment, the usual clonic and tonic convulsions, involving the fore limbs only, followed the injection of the drug, but the blood pressure usually fell. Moreover, the minimal dose necessary to elicit the convulsion was, in general, lower than in control animals: death occurred earlier, and at a lower dosage. The animal could withstand fewer successive injections than control animals. Occasionally, with transection at the level of the third thoracic vertebra, a rise of blood pressure followed either the first or second injection of the convulsant agent. In the same animal, with transection at the level of the third thoracic segment, the first injection of the convulsant agent might be followed by a fall of blood pressure during the convulsion, whereas, after the second injection, the pressure might rise. Such a sequence is shown in Fig. 4.

When the transection of the spinal cord was made at the level of the sixth thoracic segment or lower, there was a convulsion as usual, involving the fore limbs and the thoracic muscles, following the injection of absinth or camphor, and the blood pressure generally rose during the earlier responses when several successive convulsions were induced in the same animal.

4. Experiments in which the spinal cord was intact, or transected in the lower lumbar region.

The injection of camphor or absinth in minimal convulsive doses, or greater, was followed by clonic convulsions, limited to muscles whose nerve supply arose above the level of the transection in those cases in which spinal transection was done. The blood pressure, which usually fell immediately after the injection, later rose, sometimes to a great height. The maximum rise was attained during

the period of the active clonic movements, and a fall occurred rather suddenly after the cessation of these movements. In some animals, two distinct and separate series of clonic movements of the muscles occurred after the injection of a single dose of camphor monobromide. The blood pressure would rise during the first convulsion, then fall during the period when there were no clonic movements, to rise again during the second convulsion (Fig. 5). In several control animals, the minimal convulsive dose was repeated at intervals of about 15 minutes until the animal succumbed. It would withstand eight to ten successive injections, with their accompanying convulsions, or even more, before death occurred.

The Cardio-Vascular Changes After Ablation of the Cortical Motor Area.

In some acute experiments, the cortical motor area was removed, and a series of doses of camphor monobromide was administered, beginning below the minimal convulsive dose for control animals and increasing until the lethal dose was reached. This, in general, is less than for control animals. No convulsive movements of a clonic type appeared. As the dosage was increased, a few mild tonic movements appeared, culminating in tonic convulsions at the sub-lethal and lethal doses. These were less than in the controls.

While there was a considerable rise of blood pressure at the lower doses, when no convulsions appeared, there was always a fall in blood pressure during the tonic convulsion. The pulse rate, in many cases, fell with the falling blood pressure. In general, in these animals, blood pressure is lower and pulse rate higher, than in controls.

Table IV shows the relations of heart rate and blood pressure in a typical experiment.

The experimental observations show that the rise of blood pressure during a convulsion is due to the activity of the skeletal muscles, and not to any direct action upon the vaso-constrictor mechanism. The fact that in some cases a rise of pressure, and in others a fall, occurs in the phase of relaxation in the human subject shows that there is no constant vaso-constrictor element in epilepsy; and we are strongly inclined to the opinion that the rise of blood pressure, in those cases in which it occurs, is due to the action of the skeletal

muscles during the convulsion. In the absence of any general vasoconstriction, as shown by the general arterial blood pressure, during a convulsion in the human subject, we are much inclined to doubt the validity of cerebral vascular spasm as a causative factor in the genesis of a convulsion as it is encountered clinically. The difference

TABLE IV.

Camphor monobromide.	Before injection.		During reaction.		After reaction.	
	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.
1st injection (no convulsion)	120	50	102	110	120	52
2d injection (no convulsion)	138	60	150	110	138	60
3d injection (mild tonic movements)	162	76	138	110	182	80
4th injection (tonic movements)	240	98	198	66	216	65
5th injection (tonic convulsion)	264	90	198	65	216	60
6th injection (tonic convulsion)	210	78	138	50	96	20

in the blood pressure response in the two types of patients, shown in Table III and Fig. 2, seems to us to be due, as one factor, to the difference in the action of the skeletal muscles in the two types. We do not know upon what this difference in the effect of the muscular action depends, nor what other differences there may be in these patients.

Changes in Pulse Rate Under Experimental Conditions.

These changes can best be presented in tabular form in connection with the blood pressure changes which occur at the same time. There are four general relations possible. All of them may be found in a series of convulsions induced in a single animal, as in Table V. (See also Fig. 6.)

TABLE V.

(January 28, 1931.)

Camphor monobromide.	Before injection.		During convulsion.		After convulsion.	
	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.
1st injection	174	128	208	90	132	138
2d injection	222	100	208	110	176	108
3d injection	180	110	204	125	156	80
4th injection	192	70	120	40	124	40

These conditions are:

1. An increase of heart rate with a fall of blood pressure during the convulsion. This is the condition which we have found most frequently during the first convulsion induced in an animal.

TABLE VI.

	Before injection.		During convulsion.		After convulsion.	
Camphor monobromide.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.
Cat No. 1.....	150	138	180	84	170	80
Cat No. 2.....	174	128	208	90	132	138
Cat No. 3.....	150	120	192	90	164	120
Cat No. 4.....	144	110	186	85	160	105

Table VI shows the pulse rate and blood pressure before, during and after the first induced convulsion in four different animals.

2. A decrease of heart rate with a rise of blood pressure during the convulsion. This condition is frequently found in the early convulsions in animals in which a series of several convulsions is induced.

TABLE VII.

	Before injection.		During convulsion.		After convulsion.	
Camphor monobromide.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.
2d injection, cat No. 1.....	156	140	142	160	138	90
2d injection, cat No. 2.....	222	100	208	110	176	108
2d injection, cat No. 3.....	148	120	128	160	144	90
2d injection, cat No. 4.....	144	110	112	155	192	100

3. An increase of heart rate with a rise of blood pressure. This condition occurs less frequently than either the first or the second, under absinth or camphor monobromide, and is usually seen rather well along in the series of induced convulsions.

TABLE VIII.

	Before injection.		During convulsion.		After convulsion.	
Camphor monobromide.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.
Jan. 28, 1931, 3d injection...	180	110	204	125	156	80
Jan. 30, 1931, 6th convulsion.	218	30	224	60	235	40

4. Decrease of heart rate with a fall of blood pressure during the convulsion. This condition is found in the terminal convulsions of a series in any one animal.

TABLE IX.

Camphor monobromide.	Before injection.		During convulsion.		After convulsion.	
	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.	Ht. rt.	Bl. pr.
Jan. 26, 1931, 6th injection....	128	50	124	45	122	50
Jan. 28, 1931, 4th injection....	192	70	120	40	124	40
Jan. 29, 1931, 6th injection....	120	50	118	45	120	54
Jan. 30, 1931, 5th injection....	216	38	204	30	126	15

The Graphic Representation of Cardio-Vascular Changes in Convulsions of Experimental Origin.

When the four types of changes of blood pressure and heart rate encountered in the experimental animals are plotted, after the manner of Fig. 2, we have what at first sight appears to be a mass of confused and even conflicting results. In Fig. 6, the pulse rate is given on the vertical line at the left of the figure and the blood pressure on the horizontal line at the bottom. The extent of the cardio-vascular change in any given case is indicated by the extent of the line the smooth end of which begins at the point on the diagram which corresponds to the blood pressure and heart rate in the preconvulsive period and the arrow of which indicates the blood pressure and heart rate in the convulsive period. A long line means a considerable change in blood pressure or heart rate, or both. The direction of the line indicates the direction of change. The slope of the line indicates the rate of change. A line sloping downward and to the left indicates a fall of both blood pressure and heart rate, while a line sloping upward and to the right, indicates a rise of both these quantities. A line sloping upward and to the left, indicates a rise in heart rate with falling blood pressure, while a line sloping down and to the right, indicates a falling heart rate with increasing blood pressure. These points are shown in the quadrants I, II, III and IV of the small insert of Fig. 9. The symbols are the same in Figs. 6 and 7. The data for the construction of this diagram are taken from Tables IV, V, VI, VII, VIII and IX.

A study of the figure shows that some of the lines are very nearly vertical, indicating that the change of pulse rate is relatively much

greater than the change of blood pressure. The more nearly horizontal the lines become, the greater the relative change of blood pressure as compared with the change in heart rate. The more nearly vertical lines would indicate, therefore, cardiac change without much vaso-motor change, and the more nearly horizontal lines would indicate vaso-motor change in greater degree than cardiac. The graphic representation brings out clearly the mechanism or mechanisms involved in a change of any given kind, together with the magnitude of this change. Reference to the diagram shows that both the magnitude and the nature of the cardio-vascular change vary markedly with the condition of the animal. For example, considering the decrease of heart rate with a fall of blood pressure as given in Table IX, in which the arrow points downward and to the left, we find that the lines are all relatively short. The cardio-vascular and other mechanisms of such animals, are no longer capable of effecting changes of greater magnitude, whereas, if we consider the conditions shown in Table VI, in which the lines slope upward and to the left, we find that the lines are longer. This is particularly the case with those lines which indicate the changes shown in Table VII—a decrease of heart rate with a rise of blood pressure—and which slope downward to the right. As indicated above, the type of response may change markedly in the same animal with successive convulsions.

Following bilateral cortical motor area ablation changes of heart rate and blood pressure subsequent to the injection of a moderate dose of camphor with no resultant convulsion, fall in line with the usual changes under such conditions, *i. e.*, a rise of blood pressure and a fall of heart rate. But if the dose of the convulsant agent administered is sufficient to elicit tonic effects (the only ones seen immediately after cortical motor injury) there is a fall of pressure and a marked decrease of heart rate during the convulsive period. The lines indicating these changes are long and slope downward to the left.

The Interpretation of These Changes From the Point of View of Marey's Law.

Marey's Law (Starling, 1930) expresses the general relationship between pulse rate and blood pressure in the intact organism, in response to changes in the internal conditions in the organism. In

TABLE X.

SHOWING PULSE RATE AND BLOOD PRESSURE CHANGES, AT OR NEAR THE TIME OF CONVULSION, AND TWELVE HOURS LATER. ADAPTED FROM SPRATLING, 1904, P. 272.

Age.	Hour at which attacks most apt to occur.	Hour at which blood pressure is taken.	Pressure before convulsive period.	Pulse rate at time of convulsion.	Pressure at time most remote from convulsion.	Normal pulse rate.
18	7- 9 P. M.	12 hours later	105	80	110	78
14	7- 9 "	" " "	95	102	90	96
25	7- 9 "	" " "	140	108	120	84
18	7- 9 "	" " "	115	66	120	78
26	7- 9 "	" " "	135	66	100	72
21	7- 9 "	" " "	105	90	125	66
14	7- 9 "	" " "	90	96	145	90
18	7- 9 "	" " "	100	78	100	90
15	7- 9 "	" " "	95	66	90	90
10	7- 9 "	" " "	85	96	80	108
18	7- 9 "	" " "	105	72	115	66
13	7- 9 "	" " "	110	108	105	120
39	7- 9 "	" " "	150	72	130	84
20	7- 9 "	" " "	120	90	100	122
18	7- 9 "	" " "	130	84	125	72
18	7- 9 "	" " "	160	66	115	84
28	7- 9 "	" " "	125	90	120	120
18	9-10 "	" " "	108	68	120	88
26	9-10 "	" " "	135	76	115	84
43	9-10 "	" " "	155	100	155	96
24	9-10 "	" " "	135	76	130	80
22	9-10 "	" " "	120	88	100	88
32	9-10 "	" " "	125	88	110	76
42	9-10 "	" " "	150	76	115	80
25	9-10 "	" " "	175	68	120	104
25	9-10 "	" " "	140	64	145	76
38	9-10 "	" " "	144	88	155	88
24	9-10 "	" " "	95	88	105	80
29	9-10 "	" " "	105	84	90	76
25	9-10 "	" " "	130	88	115	96
40	9-10 "	" " "	155	88	135	76
34	9-10 "	" " "	105	76	110	88
17	9-10 "	" " "	130	76	105	100

general, the heart rate falls as the blood pressure tends to rise, and conversely. It has been stated (Erlanger and Hooker, 1904) as follows:

Determinable factors.		Causative factors.	
Blood pressure.	Pulse pressure times pulse rate.	Energy of heart	Peripheral resistance.
Constant	Increased	Increased	Diminished
	Diminished	Diminished	Increased
Increased	Unchanged	Increased	Increased
	Increased	Increased	Unchanged
	Diminished	Unchanged	Increased
Diminished	Unchanged	Diminished	Diminished
	Increased	Unchanged	Diminished
	Diminished	Diminished	Unchanged

The mechanism of the changes in the heart rate with changes in peripheral resistance of blood vessels involves the afferent and efferent fibers of the depressor and vagus and the efferent fibers of the nervi accelerantes from the stellate ganglion, together with other sympathetic fibers passing across to the cardiac plexus from the thoracic ganglia below the stellate (Cannon).

The central mechanism lies in the medulla. The integrity of all parts of the extrinsic cardiac nervous mechanism is necessary for the full control of heart rate in changes of blood pressure. Moderately deep anæsthesia abolishes the cardiac response to changes of blood pressure (Wickwire). In the first two cases (Tables VI and VII) of the possible four tabulated above, Marey's Law holds; while in the third and fourth cases (Tables VIII and IX), it does not. It is noteworthy that the first two cases occur early in the series of induced convulsions, while the third and fourth cases are met with in the late and terminal stages. Referring back to Table III and Fig. 2, we find a rising pulse rate and a falling blood pressure, as in case 1 of our experimental results, in nearly one-half the cases (open circles and open squares). A little more than one-half the cases shown in Table III and Fig. 2, show a rising heart rate with a rising blood pressure. Evidently something has gone wrong with the regulatory mechanism of the heart in this group of cases. As we have stated above, we do not know the reason for this departure from Marey's Law, but it falls in line with other experimental observations which we have made during asphyxiation of the bulbar mechanism. It is the regular condition which we meet during

cerebral anæmia from occlusion of the head arteries, except in those animals in which the vagus action is unusually strong.

In the light of the experimental results and from the point of view of Marey's Law we may now examine some facts as to the cardio-vascular changes given by Spratling (1904). He measured blood pressure and pulse rate at some time near to the appearance of the convulsion—exactly at what period we cannot make out—and again 12 hours later. These results are given in Table X. On closer examination of the table, one finds that there are examples of each of the four possible combinations of changes of heart rate and blood pressure which we have found experimentally. The graphic representation of these changes (Fig. 7) further emphasizes the similarity between the clinical and the experimental findings. The conditions indicated in the second case—rising heart rate with falling blood pressure—and in the fourth case—falling heart rate with rising blood pressure—of the smaller figure at the upper right hand corner, indicate those cases which follow Marey's Law. The conditions shown in quadrants one and three indicate departure from Marey's Law.

Applying now the considerations set forth in the discussion on the graphic representation of cardio-vascular changes in convulsions of experimental origin, Fig. 7 loses some of its characteristics of confusion and becomes a source of interest and instruction. Just as the results of separate and distinct conditions were used to construct Fig. 6, so we may use the experience thus gained to disentangle the confusion of Fig. 7. We find lines sloping upward and to the left, and lines sloping downward and to the right, to be longer in general than lines sloping upward to the right or downward to the left. Furthermore, the occurrence of lines sloping downward and to the left, indicating a fall of blood pressure and heart rate, is an indication of serious impairment of the cardio-vascular system. Without access to the clinical records of such patients, one cannot say whether they survived the period of examination very long or not. It would be extremely instructive if we could learn this. The records which we have show that only one of the patients (number 6, Table III) in the cardio-vascular group has died, the cause of death being broncho-pneumonia. Taken in connection with our postmortem observations on animals, one would look for numerous small hæmorrhages in the lungs of a

patient who showed such a high blood pressure (180 mm.) during a convulsion.

A closer study of patients at the time of the convulsion, together with adequate blood pressure and pulse records, is urgently needed for further statistical study. The results of such a study might lead us farther into an understanding of the central conditions in these clinical cases. Practically none of the data found in the literature is of any value for such a study.

The question of the relative magnitude and direction of change of heart rate and blood pressure at different periods in the life of the epileptic is raised by a study of the cardio-vascular responses of the cases here reported. If the subjects (Tables III and X) are divided into different age groups of six years each, starting with 10 years and continuing up to 42 years—the age of the oldest patient on which data are available, and the changes of heart rate and blood pressure are shown graphically for each group, one gets the impression, from a study of the figures (8 to 12 inclusive) that the type and magnitude of the response change with age. In the figures, a central point, with no numerical value, is taken, from which the changes in heart rate and blood pressure are measured, as in Figs. 6 and 7. Starting at this central point, increases of heart rate in the immediate post-convulsive period, or as near to it as possible, are shown on the vertical line extending upward from the central point, and decreases of heart rate are shown on the vertical line extending downward from the central point. Similarly, increases of blood pressure in the post-convulsive period, or as near to it as the data show, are indicated on the horizontal line extending to the right from the central point, and decreases on the horizontal line extending to the left. The scale indicates changes of 10 beats a minute in the heart rate, and changes of 10 millimeters of mercury in blood pressure. In the earliest age group (Fig. 8), 10 to 16 years inclusive, the changes in heart rate and blood pressure are distributed in three quadrants, and are not particularly great in magnitude, with one exception. This is true also of the second group, in which all four quadrants are represented (Fig. 9), from 17 to 22 years, inclusive. A considerable change is noted in Fig. 10, age 23 to 28, inclusive. All four quadrants are still represented, but the magnitude of the change shown in one patient in quadrant I is greater than any that has been encountered previously. In

Fig. 11, age 29 to 34, inclusive, changes of great magnitude are also shown, with no patient represented in quadrant IV. It is in this group that the only death in our series of cardio-vascular studies has occurred (Patient No. 6, Table III). The change of blood pressure was 45 mm. of mercury (135 to 180) and the change of heart rate 64 (from 86 to 150) per minute. This is next to the greatest change shown in quadrant I. As explained previously, changes in quadrant II are in accord with Marey's Law. In Fig. 12, age group from 35 to 40, inclusive, all the changes, except one in quadrant IV, and one questionable case in quadrant III, show an increase of heart rate, and pressure changes of considerable magnitude. Since there are only two patients over 40 on whom we have any data, no chart is shown for any age groups above 40. In one patient, the change falls in quadrant IV, being in accord with Marey's Law, and the changes are not of great magnitude.

It would appear from the graphic representation of the cardio-vascular changes that there is, in general, an increase in the magnitude of the changes with advancing age of the patient, and that they tend more and more to fall in quadrants I and II. More clinical observations are needed on this point before one can positively say that there is a change in definite directions with the progress of the disease. We present the data here in the hope that others may become interested in the cardio-vascular conditions in general epilepsy.

Observations on Respiratory Changes.

CLINICAL.

We find a similar paucity of observations in the literature in regard to the respiratory variations during or after an attack; Echeverria in 1870 reported a decrease in the respiratory rate in a group of 40 patients. In 1910 Knauer made graphic records of the respiratory movements during attacks in a child with Jacksonian epilepsy. From these records it appears that at the beginning of the attacks there was a complete cessation of the respiratory movements. Later, there was a considerable increase in the depth of the respiratory movements, and after an attack, the breathing became irregular, occasionally stopped entirely, or at other times simulated the Cheyne-Stokes type. Lennox has made numerous graphic

records of the respiratory movements in epileptics and stated that they show marked irregularity in the rate and depth of breathing. Apparently these records were made during the interparoxysmal periods. He and Cobb further state that "during petit mal there was either no change or an increased depth of respiration."

In our own observations we have found a respiratory standstill during the tonic phase of the convulsion; in the clonic phase we were able to observe in a few instances an increased respiratory rate, and immediately after the cessation of the clonic movements a decrease of 50 per cent in the rate.

We were able to make a more accurate observation in a case with petit mal attacks, while the patient was undergoing a basal metabolism test and was breathing into the oxygen tank of the Benedict-Roth apparatus. During the first test (Fig. 13), the average respiratory rate was about 17 to the minute. During the second test, five minutes later, there was an increase in the respiratory rate to 21 in the first and second minutes. In the third minute, the patient had an attack during which she became pale, began to roll her eyes and toss around. Her respiration at that time decreased in frequency to 15 a minute. In the fourth minute of the test the rate was 14, with irregular movements of increased depth. In the fifth minute, there were only eight respiratory movements in almost three-fourths of a minute, while the depth of the breathing was almost doubled. At this point the patient became confused, threw out the mouth-piece connecting with the apparatus and jumped out of bed, so that the observation could not be continued. It is interesting to note, as can be seen from Fig. 13, that the basal metabolism in the second reading during the attack was considerably increased as compared with that of the first reading.

EXPERIMENTAL.

The intravenous injection of sub-convulsive doses of absinth or camphor monobromide is followed by acceleration of the respiratory rhythm for a period of three to seven minutes or even longer; as the dose is increased, there is first, a sudden start on the part of the animal, perhaps a look of bewilderment or surprise and then a few isolated twitches about the head and face, perhaps of the fore limbs and sometimes of the hind limbs, but no sustained convulsion.

The acceleration of the respiratory rhythm persists for some time after all these occasional twitches have disappeared. As the dose is still further increased, the isolated twitches merge into a sustained and continuous series of clonic manifestations, during which it is difficult to make out the individual respiratory movements. The tracing of respiratory movements becomes more or less of a confused record of muscular movement, usually considerably greater in amplitude than the respiratory movements which preceded the convulsion. As the violence of the clonic movement dies down, the accelerated respiratory rhythm again becomes apparent, and the animal may lie as if exhausted, with the mouth open and the thorax rising and falling rapidly. This stage of hyperpnea gradually passes and the animal becomes more alert as the interval from the last dose of the convulsant agent lengthens. When the dose of the convulsant agent is still further increased, approaching the level of the lethal dose, there is generally a cry or moan, long drawn out as the air from the lungs seems forcibly expelled through the closed glottis. There may be two or three such cries in succession as tonic extension of all the limbs and opisthotonus come on. If the dose is not quite lethal, this tonic phase may be followed by a clonic as the effect of the drug seems to wear off a bit. The acceleration of the respiratory rhythm following such a heavy dose is not so noticeable as that following less massive doses. As the lethal point is reached, the clonic phase following the initial tonic contractions may be followed, in its turn, by a slow failure of respiration and the death of the animal in 10 to 15 minutes. If the dose is a little larger, the initial tonic phase following the injection is succeeded by a gradual relaxation of all the limbs without any clonic convulsions and without acceleration of the respiratory rhythm. Death may occur in from one to three minutes as compared with the longer interval when clonic movements follow the tonic.

In some control animals, in which bilateral vagotomy had been done under general anæsthesia one or more hours previous to the injection of the absinth, there was some respiratory acceleration following the injection of the absinth, but we have never seen the rapid, panting type such as occurs in animals without lesion of the vagi. After vagotomy, with the lower dosage of the convulsant agent, the clonic movements of the limbs may be intermitted for a

time while several respiratory movements occur, after which the clonic movements are resumed.

Ablation of the cortical motor areas under general anæsthesia a few hours before the injection of the convulsant agent increases the dose of drug necessary to elicit a convulsion and this is tonic when it first appears. The acceleration of the respiratory rhythm occurs at or below the mean minimal convulsive dose for a control animal.

Muskens (1929, p. 4) quotes Weiss's observation (1882) to the effect that, in a patient with Cheyne-Stokes respiration, the convulsions occurred in the intervals between the groups of respiratory movements and not at the time of such movements. He argues that the convulsions appear at the time when there is the least accumulation of carbon dioxide in the blood and tissues, or when the asphyxiation is least. In some animals, observed by one of us (P.) some years ago, it was noticed that after severe injuries to, or extirpations above, the medulla, and other procedures, which eventually resulted in a low blood pressure, the pulse rate would gradually fall until just before a respiratory gasp there would be a rather sharp dip in the blood pressure curve corresponding to the very marked slowing of the heart. There was an equally sharp rise after the first respiratory gasp and a resumption of the higher cardiac rate. Shortly after the last gasp of such a series, the heart rate would again slow down, and the whole cycle would be repeated. The greatest accumulation of carbon dioxide in the system occurred just before the first respiratory movement of the group.

A similar condition was observed in a post-encephalitic patient. In Fig. 14, the broad upper line was made by the writing point of a tambour attached to a stethograph about the chest. The electrocardiogram is shown below. It will be observed in the figure that the heart rate is highest just after, or coincidently with, a respiratory movement, and that this rate gradually slows until the next respiratory movement occurs. Both of the above observations suggest strongly that there is a continuous increase in the concentration of carbon dioxide in the interval between the groups of respiratory movements. Such an increase is to be expected if the metabolism of the animal or patient continues at any uniform rate during the interval between the respiratory groups.

The left phrenic nerve passes down through the thorax so close to the heart that it often lies in contact with the pericardium. The action current of the heart is of sufficient magnitude to be led off to a galvanometer. Medical students are familiar with the experiment of laying the nerve of an excised muscle-nerve preparation of the frog upon the exposed heart of a mammal, *e. g.*, cat, and observing the contractions of the muscle. What is not emphasized in this experiment, is that the cat's own phrenic nerve has been lying in contact with the pericardium for some months or years, and has only rarely, or perhaps, never, been stimulated by the action current of the heart. What is not emphasized in the experiment, is that only hyperexcitable nerves—nerves in which, according to the Ritter-Valli law, the wave of increased excitability beginning at the cut end of the nerve and proceeding peripherally, has appeared following the anatomical division of the nerve—are stimulated by the action current of the heart. On the other hand, the twitching of the left side of the diaphragm with the beat of the heart is frequently noted in experiments on the occlusion of the head arteries, particularly if the occlusion be a long one, and it occurs frequently in any condition in which for any reason the internal mammary artery and other branches from the carotid and subclavian arteries are ligated (Pike, 1916). Twitching of the left side of the diaphragm, synchronously with the heart beat is observed rather often under absinth or camphor monobromide, particularly toward the terminal stages of the experiment. Its appearance is indicative of the gravity of the general situation, and is nearly always followed rather soon by the death of the animal. Absinth and camphor monobromide seem in some way to affect the threshold of stimulation of the phrenic and possibly of other, peripheral nerves. The condition is readily recognizable upon the graphic record of the respiratory movements.

Pupillary Signs.

CLINICAL.

With regard to the pupillary signs, we have not as yet observed a case with persistence of the light reaction during the convulsion, as Binswanger and Féré did. In both the tonic and clonic phases, the pupils are widely dilated and fixed to light. In the early stage

of the relaxation, they are still dilated, frequently unequal, sometimes fixed to light, but more frequently showing a sluggish reaction. In the third minute of the relaxation, the pupils become smaller and sometimes pin-point, showing a dilation followed by an immediate contraction to strong light. Occasionally one sees a hippus reaction during the stage of relaxation. Déjerine (1914) mentions the occurrence of a hippus in epilepsy. Usually in three to four minutes the pupils return to normal size and react well to light, at a time when the Babinski sign is strongly positive and while blood pressure and pulse rate are still high.

Lennox and Cobb remark that in an epileptic with a Horner's syndrome, the condition was aggravated during a convulsion.

EXPERIMENTAL.

The pupil dilates before the onset of the clonic movements under absinth or camphor monobromide, and we have never seen a really sustained convulsion occur without the pupillary changes. An electric light bulb, suddenly flashed in front of the eyes, fails to produce any narrowing. The rigidity of the pupil to light passes off very quickly after the cessation of the clonic movements, or may even be absent if the convulsion is not severe. The corneal reflex is sometimes present when the pupil is insensitive to light. As larger doses of the convulsant drug are given, and the tonic manifestations appear, the pupils are usually narrow immediately after the failure of the tonic extension and begin to dilate only when the respiratory failure from a lethal dose becomes apparent. The third cranial nerve may be active at a time when the central motor neurones are no longer reactive to absinth.

When the cervical sympathetic of one side is divided under ether some hours before the injection of absinth, the pupil of that side remains narrow, or may even contract more, during the time of the clonic movements, while the opposite pupil dilates as usual.

Irregular deportment of the pupils is sometimes observed, and at certain stages during the experimental convulsions the pupils may be narrowed to slits even though the light is relatively dim. The experimental findings indicate a direct excitatory action of the convulsant drug upon some part of the pupillary mechanism. Ordinarily, the influence of the cervical sympathetic predominates,

but occasionally, in animals without obvious lesion of the central or peripheral system, the third cranial may predominate for brief intervals.

After Ablation of the Cortical Motor Area.

Following the injection of doses of the convulsant agent in cats from which both motor areas have been removed, the pupillary response is somewhat altered from that in the control, *i. e.*, the pupils are more sluggish in response and neither narrow nor widen to so great an extent. The pupils are often insensitive to light, or very sluggish, at a time when no convulsion appears. When the convulsion is present, they do not respond to light, and do not widen quite as much as in the controls.

GENERAL COMMENT.

The combined clinical and experimental observations show that there is an actual excitatory process occurring coincidently with the convulsion and, as we believe, standing in a causal relation to the events occurring in such a convulsion. The violent responses of the skeletal musculature, the cardio-vascular changes, the alterations of the respiratory rhythm and the pupillary phenomena seem to us to be dependent upon an actual excitation of the cells of origin of efferent fibers. There is another problem associated with this, namely, whether or not all the manifestations of a convulsion can be accounted for on the basis of cortical or higher motor neurone excitation, or whether one must go well below the cortex in order to elicit some of the reactions occurring in an epileptic seizure. No categorical answer can be given at this time, or at least, we cannot give such a categorical answer and we would be extremely skeptical of the integrity of such a categorical answer based on any of the known facts. More work, both along the line of clinical observation and experimental analysis, seems to us necessary to answer this question or these questions. One part of the inquiry might be phrased in a slightly different way and would then become: what is the lowest level in the central nervous system which is affected directly by the agent or agents giving rise to the convulsion?

The appearance of the Babinski reversal in the post-convulsive period indicates that there is either a failure of function of the cells

of origin of the pyramidal tract or a block of conduction of nervous impulses somewhere along this tract. If one accepts the view that the Babinski reversal requires the participation of the so-called extra-pyramidal tract for its genesis, the conditions which have brought about the failure of the pyramidal tract cannot have extended as low as the lowest cells of origin of the fibers of the extra-pyramidal tracts. Before one can assign a lower limit to the extent of the direct action of the conditions leading to the convulsion, it would be necessary for someone to make a categorical statement of the lowest level at which cells responsible for the Babinski reversal may lie. Further discussion without more facts appears fruitless.

The rise of blood pressure and the changes of pulse rate may be accounted for on the basis of cortical excitation (Francois-Franck and Pitres). The pupillary diameter is also increased during the period of high blood pressure. Viewed from a somewhat different standpoint, the irregularities of the cardio-vascular response in epileptic patients, who otherwise show such marked uniformity in the muscular and pupillary responses, would indicate that there cannot be the same uniformity of action as far down as the cells of origin of the cardiac and vaso-motor mechanisms in the medulla. Differently stated, the fact that one may find four different types of cardio-vascular response in epileptic patients, as well as under experimental conditions, seems inconsistent with a uniform action upon the central cardio-vascular mechanism itself. Two of the four possible cases are in accord with Marey's Law, while the other two indicate a departure from it.

We might postulate, as a corollary, that the great uniformity of response in certain phases of the convulsive manifestation, and the wide variety of response in other phases seem incompatible with the view of a psychogenic origin of epilepsy in patients who would not know what particular types of response they should choose for their own.

The general considerations on the condition of the peripheral blood vessels under absinth or camphor monobromide, and the rôle of the skeletal musculature in the changes of blood pressure in convulsions of experimental origin have been given in detail elsewhere (Coombs and Pike, 1931) and need not be repeated here. We may say that, on the basis of the experimental results, we

look upon the blood pressure changes in general epilepsy as something due to the action of the skeletal musculature rather than as something due to any primary action upon the vascular nervous mechanism.

Some of the facts adduced in this study suggest a progressive change in such conditions as the duration of the Babinski reversal and the cardio-vascular conditions, and that the rate of change of these phenomena may bear some relation to the prognosis and expectation of life in the case of particular patients. More study is required before a definite answer can be given to any of the questions arising from these suggestions. The surest method of getting an answer would be the careful study of the life history of a number of patients suffering from epilepsy from the time the disease is first diagnosed until the exitus of these patients. Such a method would require time, and for immediate purposes some other method would seem advisable. The statistical method is the one which first comes to mind. By its use, we might get more light, not only on the phenomena of a convulsive seizure at any given age, or the general conditions in patients at any given age, but also on the other question of how and to what degree or at what rate the changes occur in the course of the disease from its inception to its termination after a period of years. The task of the experimentalist is the analysis by his own peculiar methods of something which is observed, and he can be sure of what he is to analyze only when he knows what the facts are. At present, the experimental attack on epilepsy is handicapped by a lack of knowledge of what actually occurs clinically. The experimentalist reasons backward from the phenomena which he is expected to reproduce to the conditions which produce them. There seem to be in epileptic patients a number of conditions which the experimentalist is inclined to consider the effects of the disease rather than its cause. He forms an idea of a possible cause only when he can get some adequate idea of the nature and magnitude of the effects which are produced in a time which may be more or less definite; and the order in which things occur may be a very significant guide in the process of arriving at an idea of the cause.

It has long been recognized by students of the more general aspects of physiology, *e. g.,* Jost (1908) in his lectures on plant physiology, that changes of matter and energy underlie all other

changes of whatsoever kind in all living organisms. The physiologist finds two questions demanding an answer in every activity or manifestation of activity which it may fall to his lot to study. One is, "What material or what stuctures enter into this particular manifestation of activity?" and the other is, "What is the source of the energy which gives rise to it?" More specifically in the investigation of epilepsy the questions become, "What structures are concerned in the production of the convulsions?"—the problem of localization—and "What kind of a process or physical agent gives rise to them?"—the problem of the cause of epilepsy. We incline to the view that the cause is anatomical in nature.

GENERAL SUMMARY.

A number of investigators have studied the Babinski phenomenon in convulsive states; only a few give accurate data as to the time of its appearance, its uni- or bi-laterality, or its duration.

In our own observations of convulsive attacks in 69 epileptics the Babinski sign on plantar stimulation was positive in 65 instances—*i. e.*, 94.2 per cent, after cessation of the clonic movements.

In 37 observations—56.9 per cent—the Babinski sign became positive immediately after the last convulsive movement.

In 28 instances—43.1 per cent—the Babinski sign became positive two or three minutes after cessation of the convulsion. During this lapse of two or three minutes, there was no response to plantar stimulation, or, occasionally, even a flexor type of response was observed.

In 17 observations—26.1 per cent—the Babinski sign could be elicited on one side only; in the other 48 cases—73.9 per cent—it was elicited on both sides.

The duration of the Babinski sign is from half a minute to 25 minutes. The graph of the frequency of occurrence of any given duration, plotted against the duration, shows two very distinct and completely separated maxima at one minute and at three minutes, with a less distinct maximum at five minutes, and scattered cases up to 25 minutes. The duration of the sign appears to increase with the age of the patient.

The mechanism of the Babinski sign is still in the realm of speculation. The most we can say from our own observations is

that they suggest a transitory derangement of the pyramidal system with a consequent appearance of pyramidal tract signs of a fleeting character.

The pulse during the convulsive seizures is obscured by the muscular movements in both the tonic and clonic phases, and the blood pressure cannot be measured, for the same reason. Immediately after the cessation of the convulsion there is a pronounced tachycardia with a gradual decrease in pulse rate until the recovery of the interparoxysmal rhythm in 5 to 30 minutes.

In our clinical observations the systolic blood pressure rose immediately after the convulsive seizure in about half the cases, and in the other half it decreased. The diastolic pressure remains about normal in the cases where the systolic pressure is increased after the convulsion, but decreases markedly with a decrease in systolic pressure.

The experimental results show a rise of blood pressure and generally of heart rate in intact, unanæsthetized animals after the intravenous injection of absinth or camphor monobromide in sufficient doses to elicit a well-marked convulsion. Neither the increase of blood pressure nor of heart rate is invariable. Of the four possible conditions of concomitant variation of heart rate and blood pressure, all have been found experimentally. There may be (1) a rise of both heart rate and blood pressure, (2) a rise of heart rate and a fall of blood pressure, (3) a fall of both heart rate and blood pressure, or (4) a fall of heart rate with a rise of blood pressure.

The same four possible types of variations of heart rate and blood pressure during a convulsion occur also in human beings. In the earlier decades of life all four types of response occur, but above the age of 30, the responses occur almost exclusively in groups 1 and 2 of the four types given above. In general, also, the magnitude of the change, as indicated by the lengthening of the lines in Fig. 7, and the data drawn from Fig. 2, tends to increase with the advancing age of the patient. (See Figs. 8 to 12.)

The fact that no increase of blood pressure occurs in experimental animals after the injection of absinth or camphor monobromide when the animal is etherized, when the skeletal muscles are eliminated by the intravenous injection of curare, or when the spinal cord is transected high in the thoracic region, shows that such rises of blood pressure as do occur in other animals are due to the action of the striated musculature during a clonic convulsion.

A similar rise of blood pressure does not occur when the convulsant agent is injected within a few hours after bilateral ablation of the cortical motor areas; the convulsions under these conditions are tonic. Nor does the rise of blood pressure occur in control animals when the dose of the convulsant agent is such that a tonic and not a clonic convulsion appears.

The wide variability of cardio-vascular changes in the human subject during epilepsy indicates a lack of any constant or specific primary action upon the cardio-vascular mechanism. Neither the experimental nor the clinical data indicate a cerebral vascular spasm as the cause of the convulsion. The physiologist has difficulty, amounting to inability, in recognizing in such a diversity of cardio-vascular reactions, any common element which would seem to be a causative factor in such a uniform motor manifestation as a grand mal seizure. In those cases in which a rise of pressure occurs, its measurement becomes possible only when, in all probability, the height of the pressure has been passed and has already begun to fall with the cessation of the clonic movements. The measurements in the experimental animals show that the greatest height of blood pressure occurs during the period of the most violent convulsions and that a prompt fall follows the cessation of the clonic movements.

One observation on the respiratory movements during a petit mal attack was recorded graphically during a test of basal metabolism. Preceding the attack there was an increase in the respiratory rate, followed by a decrease below the interparoxysmal rate during the seizure. In this phase, the amplitude of the respiratory movements increased as compared with the preparoxysmal period, and toward the end of the convulsion was twice as great. The basal metabolic rate was increased about 15 points during the convulsion.

Under experimental conditions, there is usually an increase in the respiratory rate at subconvulsive doses of the convulsant agent. When the convulsive level is reached, the graphic record of the respiration is overlaid and obscured by the clonic movements of the animal. After sub-lethal doses, the subsidence of the convulsive movements is followed by a period of rapid respiratory movements. When the dosage reaches the value at which tonic phenomena are elicited, the respiration is slowed, and may even be intermitted during the period of greatest tonic activity. Failure of the respiration follows the lethal dose, while the heart continues to beat for 10 or 20 minutes.

The respiratory changes, especially the acceleration, are in general less marked than in animals without lesions after the injection of absinth or camphor monobromide.

The pupils are dilated and rigid to light during both phases of the convulsion. During the early phase of relaxation they may be unequal and react sluggishly to light. In the second or third minutes of relaxation they may become pin-point, giving paradoxical reaction to light. The reaction of the pupil to light returns before the Babinski sign disappears, and before the pulse rate and blood pressure return to normal.

Under experimental conditions, there is also dilation and rigidity of the pupils to light, but the corneal reflex may be present at times while the pupil is still rigid.

When one cervical sympathetic nerve is divided some hours before the experiment, the pupil on the same side contracts under absinth, while the other dilates.

The usual response of the pupil to convulsant agents was decreased when the cortical motor areas were removed a few hours before.

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DESCRIPTION OF FIGURES.

FIG. 1.—Duration of the Babinski reversal in patients of various ages during the phase of relaxation following a convulsion. The age of the patient at the time of examination is given in the vertical column at the left. The duration of the sign in minutes is given along the horizontal line at the bottom of the figure. The solid black circles indicate that the patient died in convulsions. The circles which are half filled in indicate that the patient died of some pulmonary disorder. The open circles indicate that the patient is alive at the time of writing.

FIG. 2.—The changes of blood pressure and heart rate in the phase of relaxation following a convulsion, as compared with the interparoxysmal period. The pulse rate is given in the vertical line at the left, and the blood pressure on the horizontal line at the bottom of the figure. The conditions during the interparoxysmal period are shown by the circles, and in the phase of relaxation by the squares. The solid black circles and squares indicate those patients in which an increase of blood pressure and pulse rate occurred during the convulsion. The open circles and squares indicate those patients in which there was a fall of blood pressure, but an increased pulse rate during the convulsion. The black circle and square marked with a + sign indicate death from bronchopneumonia since the date of examination.

FIG. 3.—The blood pressure response to intravenous injection of camphor monobromide in an etherized cat. The blood pressure fell promptly after the injection, and respiration (not shown) failed. The recovery of the animal was spontaneous after artificial respiration was begun (shown at the right of the blood pressure drop).

FIG. 4.—Cardio-vascular response to two successive injections of equal doses of camphor monobromide. In the upper tracing, there was a fall of blood pressure. After the second injection (lower tracing) the pressure rose. The upper line in each tracing is the record of respiratory movements.

FIG. 5.—Two separate convulsive attacks resulting from the same dose of camphor monobromide. The injection was made just before the slight fall of blood pressure in the upper tracing. The top record in these tracings shows the respiratory changes. The irregularities result from the clonic movements of the skeletal musculature. The greatest height of blood pressure is attained during the most rapid clonic movements in the upper tracing. Two and one-half minutes after the injection, there was a second convulsion, shown in the lower tracing.

FIG. 6.—Chart of the changes of heart rate and blood pressure during convulsions of experimental origin in cats. The pulse rate is shown on the vertical line at the left, and the blood pressure on the horizontal line at the bottom of the figure. The direction of the change of blood pressure is indicated by the arrows. The open end of the line indicates the blood pressure and heart rate in the interparoxysmal period, and the arrow, the conditions during the convulsion. The symbols have the same significance as in Fig. 7, *q. v.* Further description in the text.

FIG. 7.—Chart showing the changes of blood pressure in the human subject from the interparoxysmal period to the period just before a convulsion. The symbols are explained on the chart. The open end of the line indicates the blood pressure and heart rate in the interparoxysmal period and the arrow the pressure and heart rate as observed at or near the convulsive period.

FIG. 8.—In this and the four following figures, the magnitude and direction of the cardio-vascular changes from the interparoxysmal period to the post-convulsive period are given for patients in different age groups. Fig. 8 shows the changes in the age group from 10 to 16 years inclusive, starting at a common point which does not correspond numerically to the actual blood pressure or pulse rate during the interparoxysmal period, but which is simply used as a point of reference, with relation to which the observed differences between the interparoxysmal and the post-convulsive values of blood pressure and heart are plotted. The significance of the different quadrants is the same as in Fig. 7. The changes of blood pressure in millimeters are indicated on the horizontal scale and the changes in heart rate in beats per minute are indicated on the vertical scale.

FIG. 9.—Shows in the same manner the changes in the age group from 17 to 22, inclusive.

FIG. 10.—Shows in the same manner the changes in the age group from 23 to 28, inclusive.

FIG. 11.—Shows in the same manner the changes in the age group from 29 to 34, inclusive.

FIG. 12.—Shows in the same manner the changes in the age group from 35 to 40, inclusive.

FIG. 13.—Shows changes in respiratory rhythm in a patient in an attack of petit mal.

FIG. 14.—Respiration and heart rate (electrocardiogram) in a post-encephalitic patient. The respiration was slow and spasmodic, indicated by the upper broad line. Two respiratory movements in quick succession are shown in the upper strip, and then no more until the two oscillations in the lower strip 10 or 15 seconds later. The slowing of the heart—nine seconds for each beat—before the respiratory movements is succeeded by a higher rate—.65 to .70 seconds for each beat immediately afterward. The heart slows again in the interval before the next respiration.

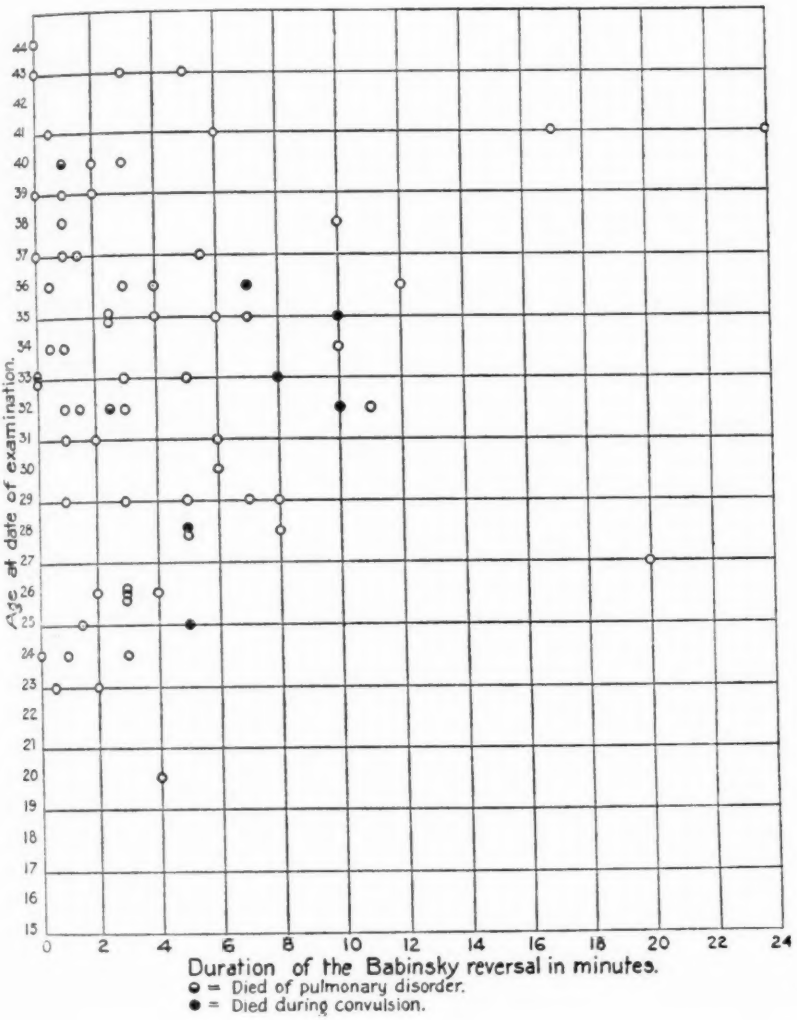
Age at date of examination.
44
43
42
41
40
39
38
37
36
35
34
33
32
31
30
29
28
27
26
25
24
23
22
21
20
19
18
17
16
15
0
2
4
6
8
10
12
14
16
18
20
22
24
Duration of the Babinsky reversal in minutes.
= Died of pulmonary disorder.
= Died during convulsion.

FIG. 1.

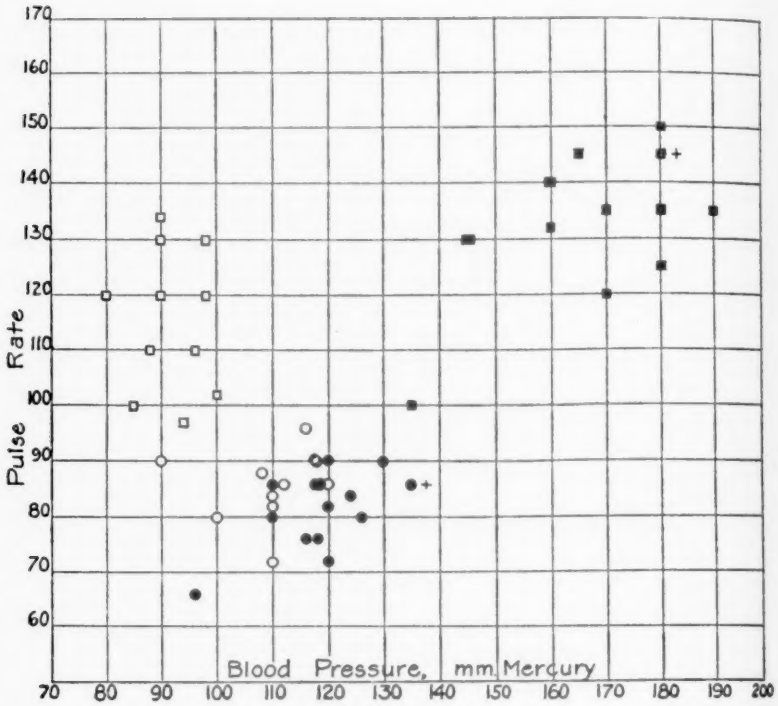
Pulse Rate
170
160
150
140
130
120
110
100
90
80
70
60
Blood Pressure, mm. Mercury
70 80 90 100 110 120 130 140 150 160 170 180 190 200

FIG. 2.

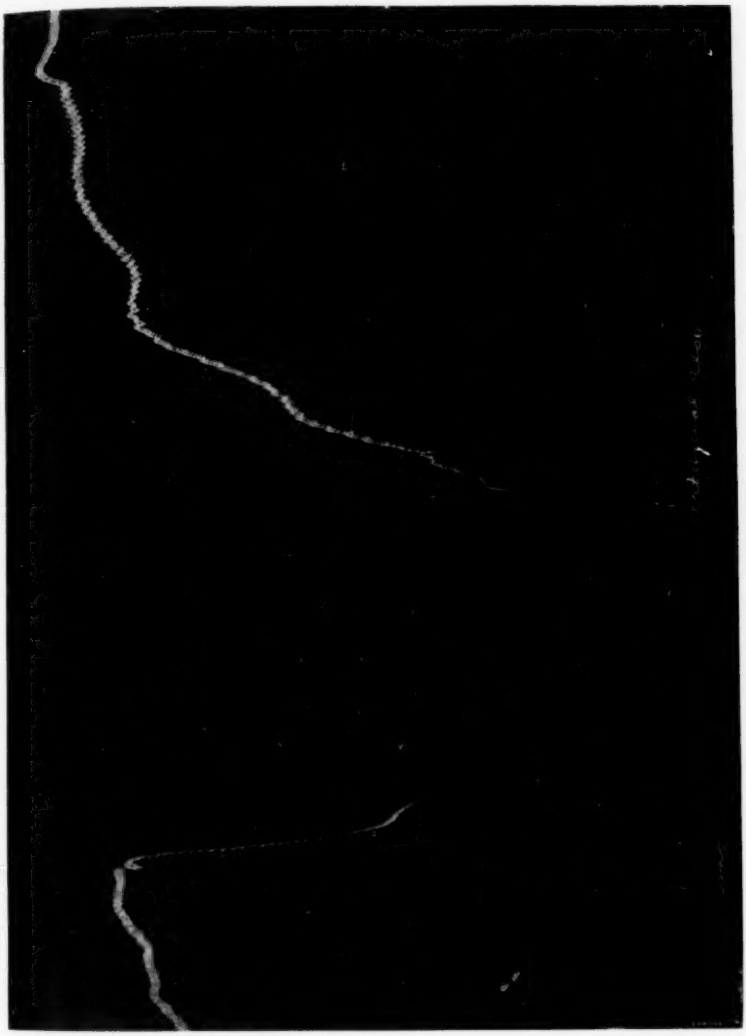

FIG. 3.

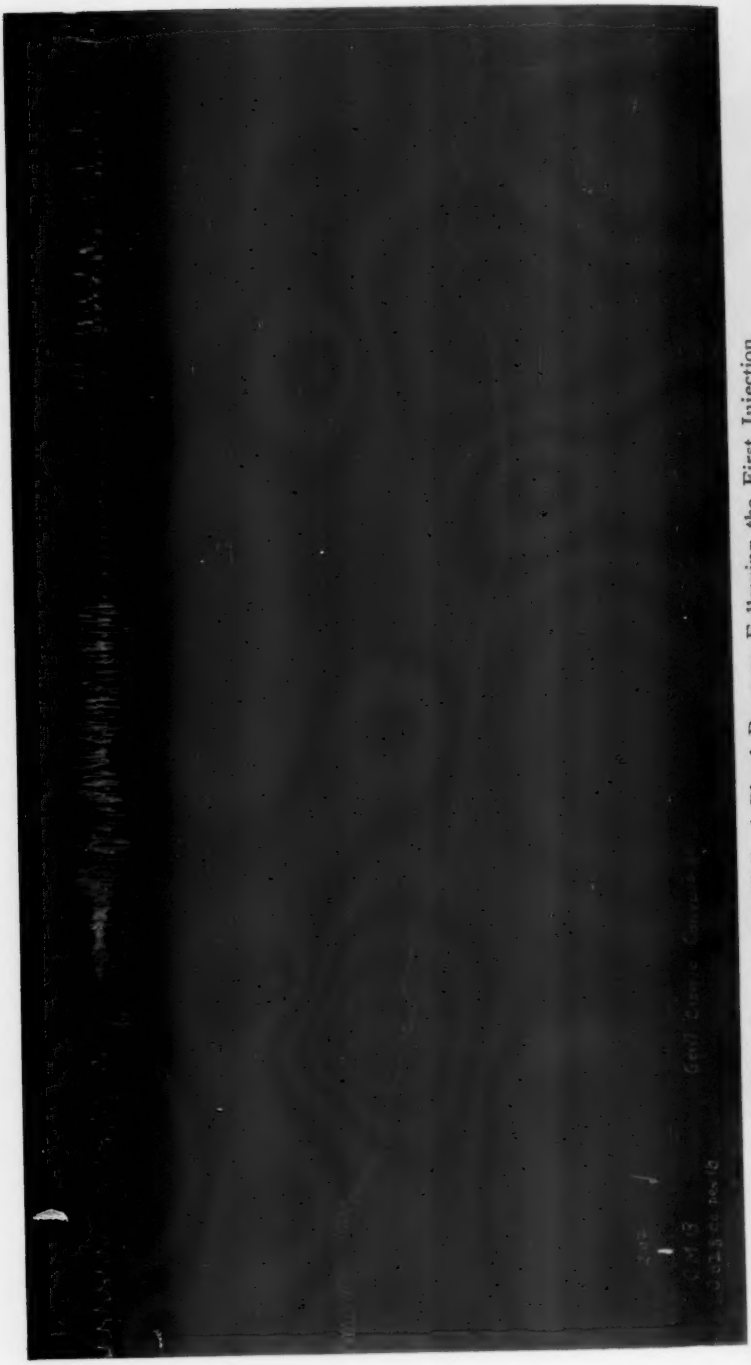

FIG. 4A.—A Fall of Blood Pressure Following the First Injection.

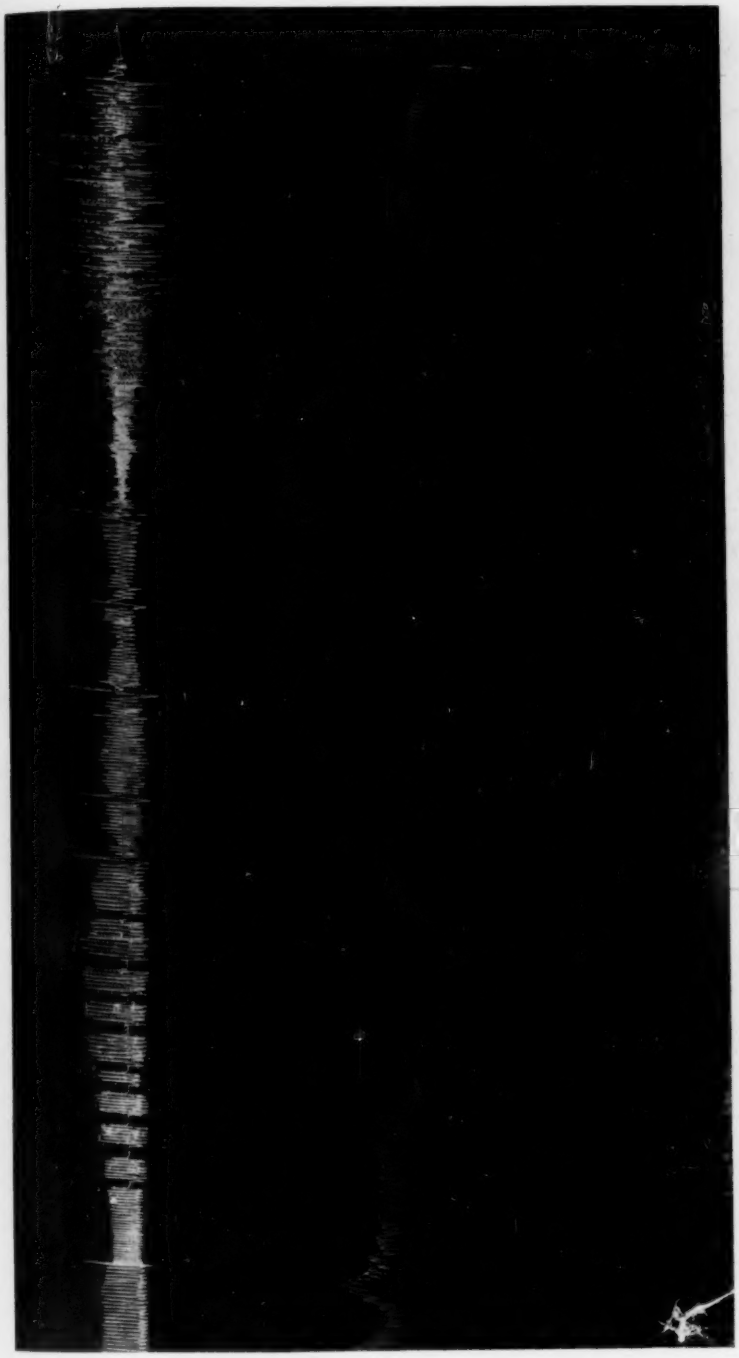

FIG. 4B.—A Rise of Blood Pressure Following the Second Injection.

FIG. 4A.—A Fall of Blood Pressure Following the First Injection.

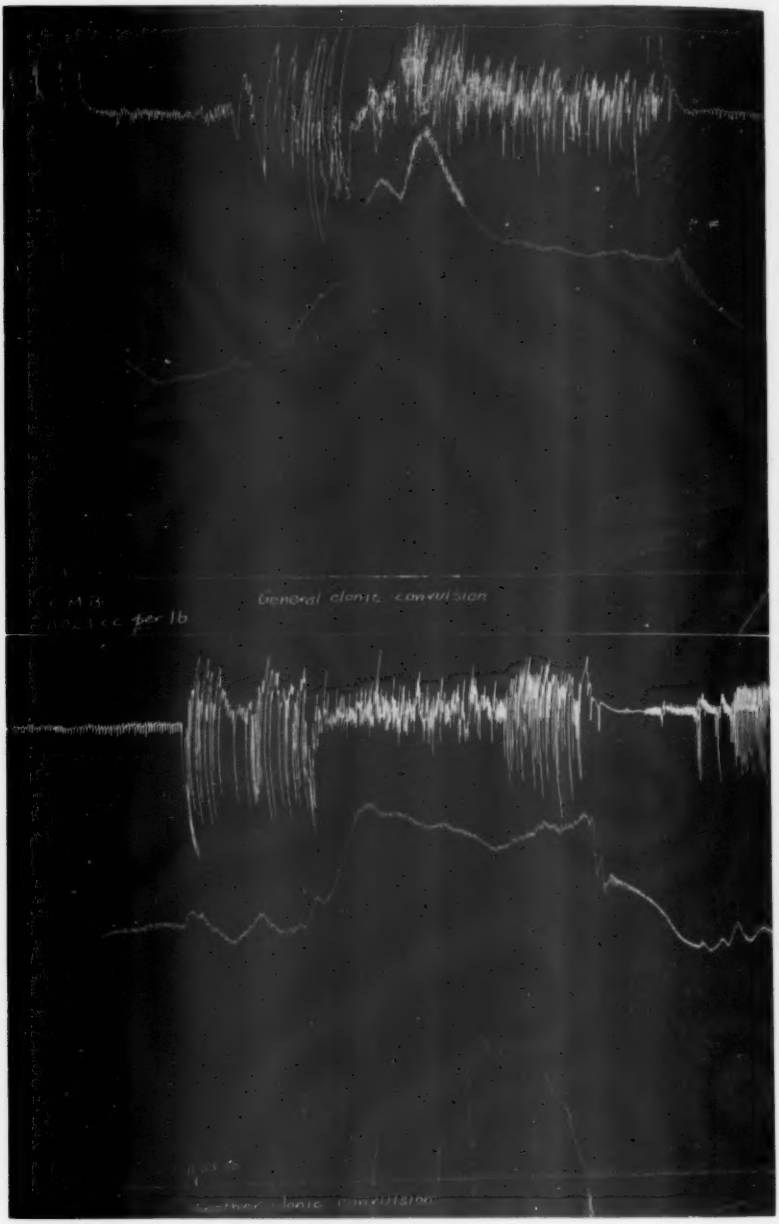

FIG. 5.—Two Successive Rises of Blood Pressure from the Same Injection of Camphor Monobromide.

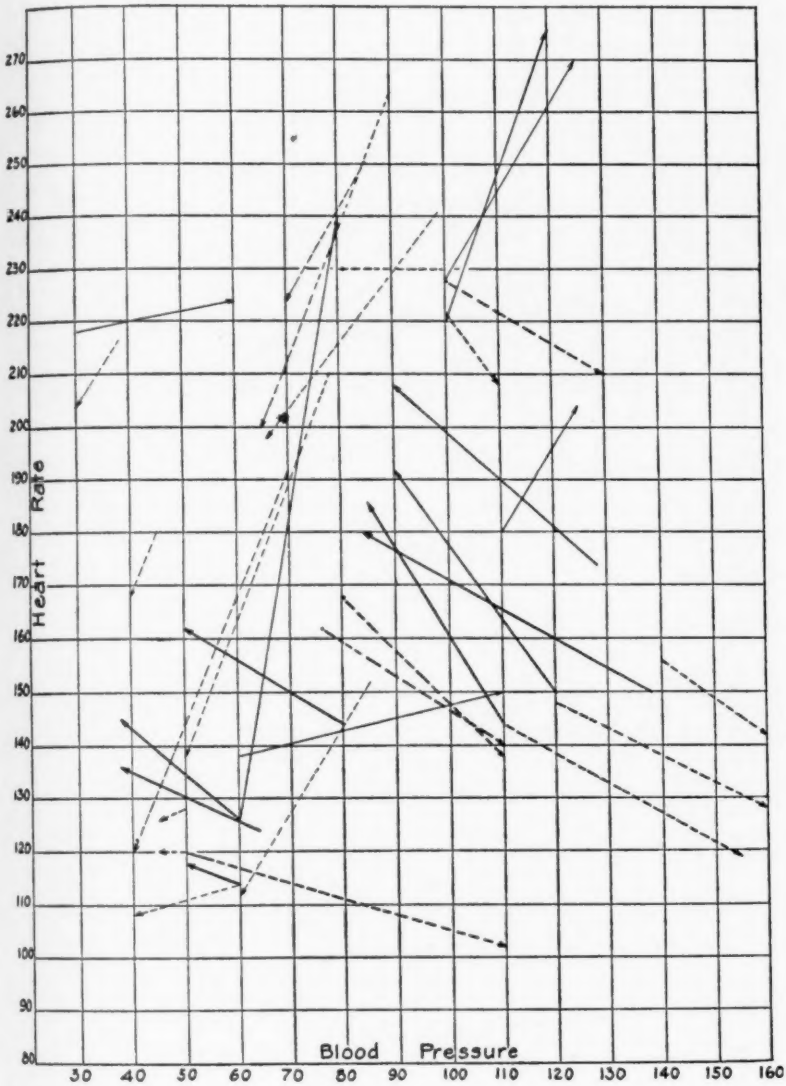
Heart Rate
270
260
250
240
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
Blood Pressure
30 40 50 60 70 80 90 100 110 120 130 140 150 160

FIG. 6.

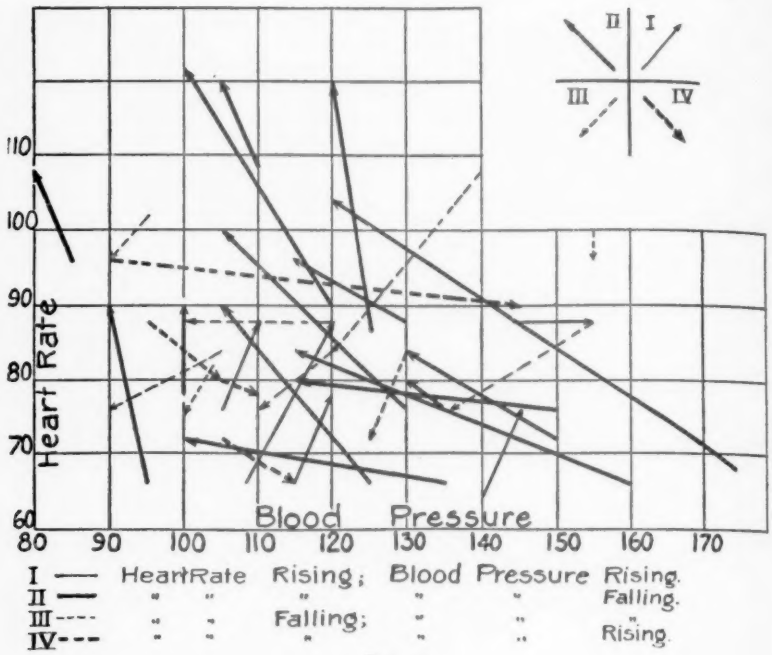
II
I
III
IV
110
100
90
80
70
60
Heart Rate
80 90 100 110 120 130 140 150 160 170
Blood Pressure
I —— Heart Rate Rising; Blood Pressure Rising.
II —— " " " " " Falling.
III ---- " " Falling; " " "
IV --- " " " " " Rising.

FIG. 7.

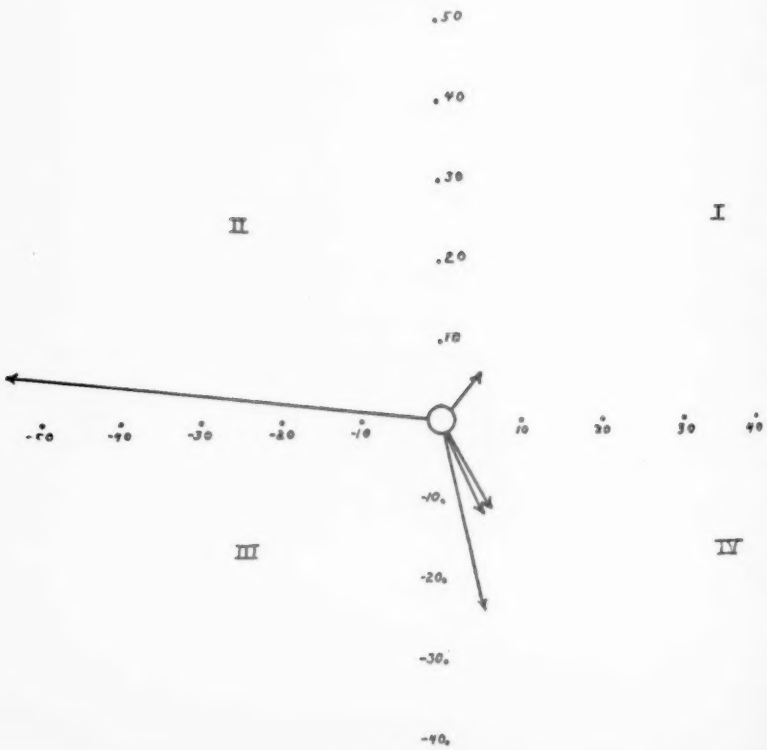
.50
.40
.30
II
I
.20
.10
-10.
III
IV
-20.
-30.
-40.

FIG. 8.

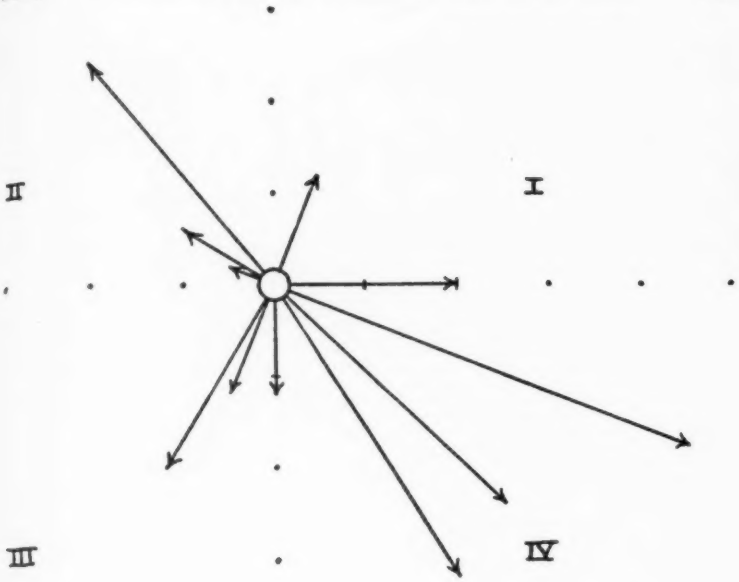

FIG. 9.

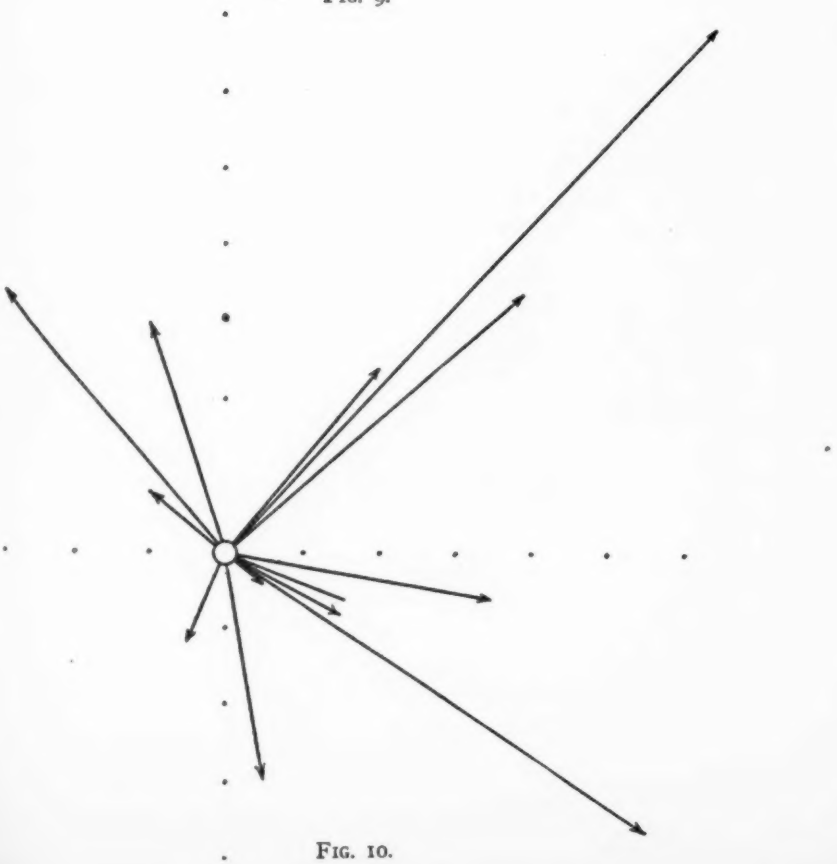

FIG. 10.

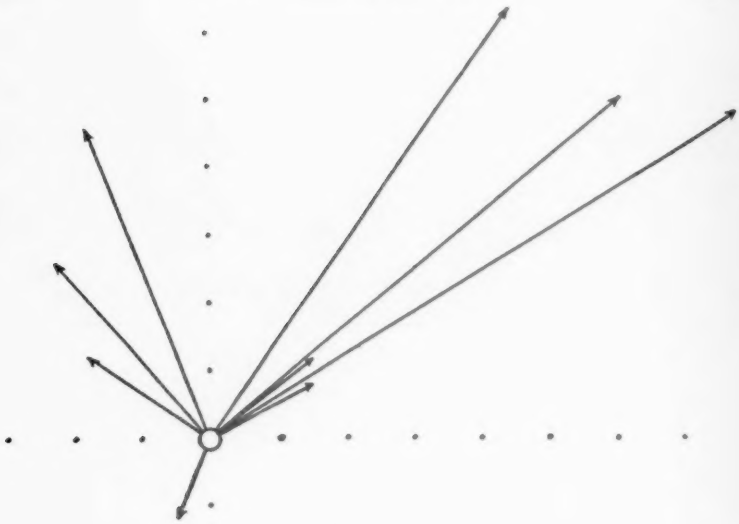

FIG. 11.

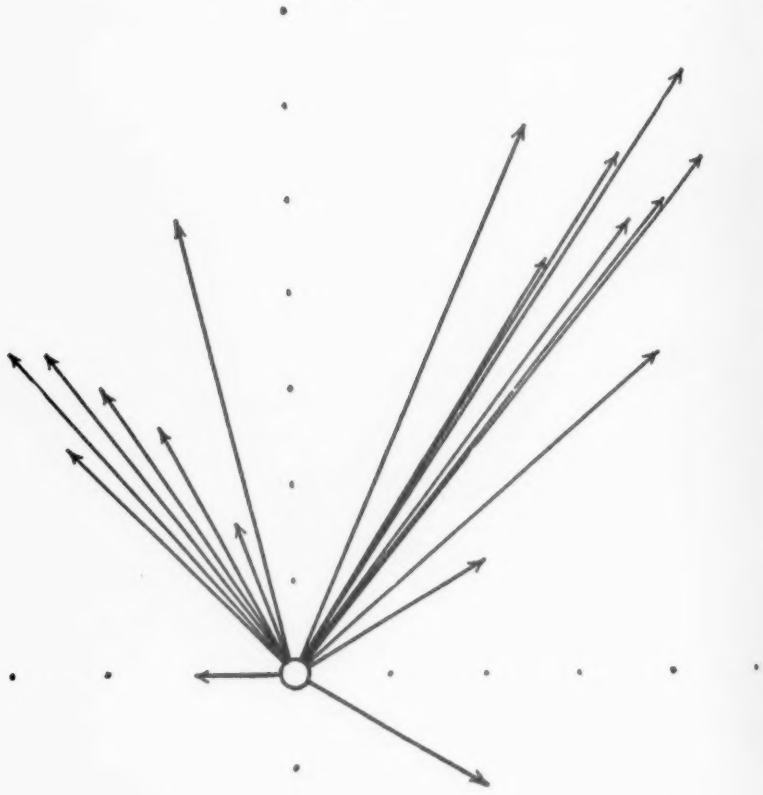

FIG. 12.

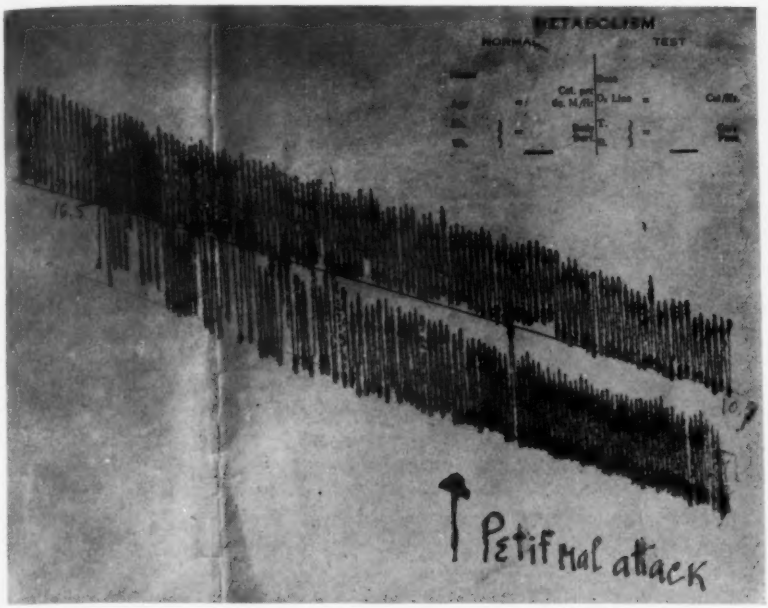

FIG. 13.

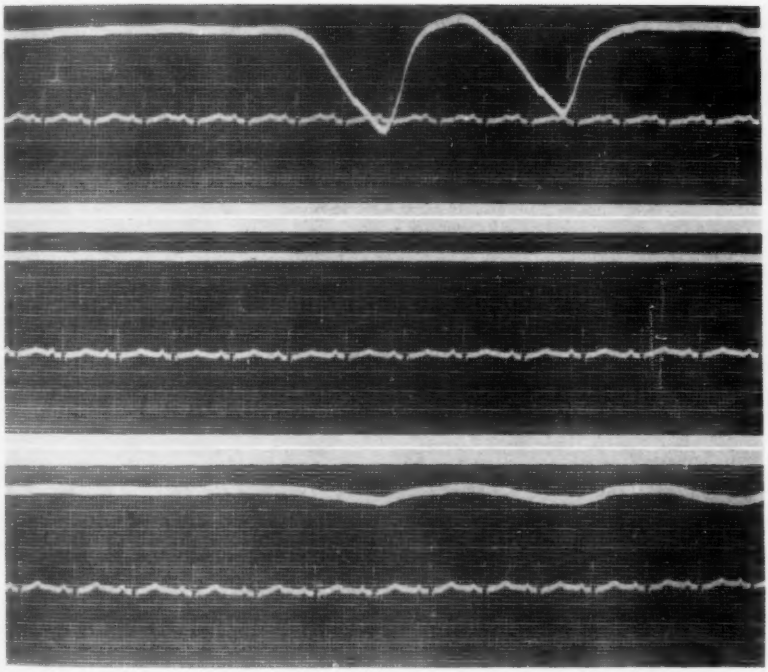

FIG. 14.—Respiration and Heart Rate in Post-Encephalitic Patient.

DISCUSSION.

Dr. S. B. Wortis (New York City).—The problems Dr. Pike has spoken of this afternoon are very important. For many years the vascular theory of epilepsy has held sway. Many people have observed brains of patients on the operating table, in the course of a convulsion. You will find descriptions of several such episodes in the literature.

That a vasoconstriction does occur, accompanying a convulsion, there can be no doubt; but whether or not this vasoconstriction is the cause of the convulsion is a question which may well be disputed.

We are coming to feel, especially in relation to work with the camphor and absinth group of convulsant drugs, that these substances induce a discharge of energy—that some of the energy so discharged may pass out over the sympathetic system and has the power to constrict vessels; that vasoconstriction of the vessels of the cerebral cortex *does* occur, but it is merely an *expression* of the epileptic fit—which has already started.

As further evidence of this fact, let me mention that if one observes the viscera of an animal during a fit, a similar vasoconstriction and subsequent vasodilatation of the vessels in the viscera is seen in many instances.

I should also like to add a word of confirmation to Dr. Pike's observation on the fall of blood pressure associated with the use of camphor and absinth. This has also been described by Muskens.

The relation of the Babinski reversal to the age groups of epileptics, and the observation that this Babinski reversal has a longer period of duration in older persons, may in some part be accounted for by differences in nerve tissue irritability, which may possibly be altered with increasing age.

Dr. J. Notkin (New York City).—May I be permitted to make a few remarks in addition to what Dr. Pike has said? The clinical details are the result of about four years' observation. I first recorded all the signs observed during the various phases in epileptic patients and then attempted to tabulate and correlate them in the hope that some light might be thrown on the mechanisms of the convulsive seizure.

It is important to note that in 69 patients the Babinski reversal was positive in 65 instances or in 94.2 per cent. In 59.9 per cent the sign was positive immediately after the last clonic movement and in the rest it was elicited 2 or 3 minutes after cessation of the convulsion. It may be of some interest that during the lapse of these few minutes there was no response to plantar stimulation and occasionally even a flexor type of response was seen. As Dr. Pike pointed out, in a certain percentage of cases (26.1 per cent) the Babinski sign could be elicited on one side, not only during the first but also during the subsequent examinations. That is a very important point, as it may indicate some difference in processes taking place in the cerebral hemispheres or in the pyramidal tracts during the convulsion.

I may add that patients over 45 years old were excluded from this report because I felt that if we included patients with arteriosclerosis or with definite organic brain condition we should obscure the problem. In fact in

older arteriosclerotic patients the Babinski sign could be elicited even two hours after the convulsion and in general paresis it was persistent for several days, whereas the average duration in the reported group was 3 to 4 minutes. We were particularly careful regarding the ages of patients in the study of the cardio-vascular signs and for this reason we excluded from our report patients of advanced ages or with a definite involvement of the cardio-vascular system. I may briefly state that during the convulsive seizure the pulse is barely perceptible and the blood pressure cannot be measured. Immediately after the cessation of the convulsion there was usually a tachycardic pulse; the blood pressure in 50 per cent of cases was considerably increased and in the other half markedly decreased. The reason for this striking variation of the blood pressure is not clear to me particularly in view of the absence of cardiac complication in our cases.

Considerable attention was paid in the past to the pulse rate and blood pressure variations immediately before a convulsion. Observations of this kind are reported in the literature but it was disheartening to sit up for hours and days with the same patient taking his pulse count and blood pressure readings at 10 minutes' intervals and never succeeding in making such observations immediately before a convulsion.

Personally, I would not care to draw any conclusions from the facts obtained. I thought that it might be of some interest to record these findings and that with accumulation of more material some conclusions may be reached at a future date.

OBSERVATIONS ON SOME VISCERAL CONDITIONS IN GENERAL EPILEPSY AND IN CONVULSIONS OF EXPERIMENTAL ORIGIN.*

By SAMUEL M. WEINGROW.

From the Department of Neurology and the Neuro-Surgical Laboratory of Columbia University.

Bethea [1] studied the gastro-intestinal tracts of 400 epileptic patients. He found dilated stomachs in 114 cases and five hour retensions in 336. Cardiospasm, pyloric spasm, gastric ulcers, defective duodenal caps and pressure deformities as well as spastic and dilated conditions of the small and large intestines were among the conditions observed. Robertson [2] x-rayed 13 epileptics and found widespread mucosal catarrh and chronic intestinal stasis. The absence of colonic stasis [3] and the presence of hypermotility in 50 per cent [4] and constipation in all of Harryman and Donaldson's cases, as well as the sensational disclosures of Reed [5, 6] in his extensive radiographic and operative gastro-intestinal work are sufficient evidence of involvement of this system in the course of the convulsive seizure. Lennox and Cobb [7] mention dilated stomachs in 5 per cent among other gastro-intestinal post-mortem findings in 423 patients. Recent studies [8] indicate that ptotic conditions are present in a large number of supposedly normal individuals. Such findings, although emphasized by previous writers,[5, 6] must, therefore, be excluded from our consideration.

Dilatation of the stomach may not be of frequent occurrence in post-mortem examinations of epileptics because death in epilepsy is usually due to intercurrent disease rather than to a particular seizure or a series of seizures. This condition has been shown to be much more prevalent during the life of the epileptic.[1]

*Read at the eighty-seventh annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Toronto, Canada, June 1, 1931.

The expenses of the experimental part of this paper were defrayed by a grant from the Commonwealth Fund to the Neurological Institute of New York.

In general medicine dilatation of the stomach presents itself in acute and chronic forms. The former is the more dangerous and the least understood. It is generally a serious complication arising in the course of some other condition, especially after operations, notably laparotomy performed for whatever cause, after abdominal injury, during acute fevers, especially lobar pneumonia, and in the course of chronic heart failure. The chronic form is due to totally different causes. One class of causes consists of involvements by pressure or pulling of the pylorus, such as are met within cicatrices of gastric and duodenal ulcer, carcinoma of the pylorus, gall bladder or head of pancreas, and those due to dilatation without obstruction, *i. e.*, atony or overdistension by gas, food or drink.[9]

In dealing with acute dilatation of the stomach Alvarez [10] states that he saw this condition develop in animals that were subjected to anesthesia for a long time. He states that Hedbloom and Cannon produced this condition in the cat by injecting croton oil into the cecum. His citation of Woodyatt and Graham's production of this condition by tying off the vessels going to the stomach, his belief that, in some cases, it is due to a powerful type of respiratory movement which filled the stomach with air, as well as his illustration of the dynamic balance between the stomach and colon [11] definitely direct us to the presence of similar conditions and factors in experimentally induced convulsions.

The genuine fit in epilepsy closely resembles that experimentally produced in animals.[12] Dilatation of the stomach is often found to be extreme on post-mortem examination of animals in which convulsions were induced experimentally.[13]

While considering the general phenomena of an experimentally induced convulsion, a study was made of the possible effects of neurophysiological, mechanical, organic, chemical and therapeutic factors upon the condition of the gastro-intestinal tract, and especially the stomach, in the course of experimentally induced convulsions. Neurosurgical procedures involving the cutting of central and peripheral structures that might be related to the functioning of the intestinal tract were considered. These consisted of removal of motor cortex, longitudinal incision of the midbrain, transverse section of the spinal cord, cutting the phrenic nerves, vagi and splanchnics, and others. Avenues of approach through

the hemic route by intravenous injection of hypertonic solutions of glucose or of tapwater, thus varying the intracranial pressure; as well as by the removal of the adrenals or injection of adrenalin for their effects upon the sympathetic system, and hence upon the stomach, were also attempted. Removal of the adrenals is sometimes associated with terminal convulsions [14] and uremia with dilatation of the stomach.[15] The latter condition is as yet but little understood, but the relationship between a rise in the urea of the blood with onset of coma and other uremic signs subsequent to dilatation of the stomach following pyloric stenosis make the possible effects of nephrectomy a suitable procedure for our consideration. In this connection the effect of ammonium carbonate seems of interest. The effects of potassium borotartrate and luminal were also observed for their action when given in association with absinth through the intravenous route.

The animals were not specially selected, but were taken in consecutive routine order from the laboratory note-book of neurosurgery at Columbia. The experiments had been done by various people. The technique of the intravenous injection of absinth has been given elsewhere. The first dose injected was usually small and below the minimal convulsive level. The dose was increased by small increments until the minimal convulsive level was passed, and the increase was continued until the lethal amount was reached. Other drugs, whose effect it was desired to study, were introduced intravenously after the minimal convulsive dose was established or administered in the food for a period of days or weeks before the injection of the absinth.

Respiration fails before the heart stops in death from absinth.

The variation in dosages and in the effects upon the gastrointestinal and cardiovascular systems, as well as some other changes occurring as a result of, or associated with, the operative procedures or administration of drugs in the experimentally induced convulsions are presented in the following tables.

Table I shows the average threshold or minimal convulsive dose per cat and per pound (columns 3 and 4), the lethal dose (columns 5 and 6) and the differences between the two quantities (5-3, 6-4) as the convulsive factor (columns 7 and 8).

It will be noted that in all the modifications or subgroups the value of the convulsive factor is less per cat and per pound than

TABLE I.

Type of experiment.	1	2	3	4	5	6	7	8	9
	No. case.	Aver. wt. per pound.	Aver. thresh.		Aver. final dose.		Aver. convul. factor		Aver. no. convulsions.
			Per cat.	Per lb.	Per cat.	Per lb.	Per cat.	Per lb.	
Controls	17	3.6	0.9	.02+ *	.20+	.05+	.21+	.03+	5.5+6
Surgical procedure	16	6.6	.15+	.02+	.28+	.05+	.13+	.02+	3.8+2
Intravenous glucose	13	5.7+	.16+	.02+	.27+	.04+	.11+	.01+	3.7+
Intravenous tapwater	8	5.7+	.16+	.02+	.18+	.03+	.01+	.00+	3+
Removal of adrenals	13	5.5	.12+	.02+	.15+	.02+	.03+	.00+	1.4
Adrenalin	4	4.6	.14+	.03+	.19+	.04+	.05+	.01+	4.5
Nephrectomy	5	5.4	.17+	.03+	.28+	.05+	.11+	.02+	3.4
Ammonium carbonate	3	6.1	.16+	.02+	.16+	.02+	.00+	.00+	4.3
Luminal	6	4.8	.10+	.02+	.21+	.04+	.10+	.02+	3
Borotartrate	3	5.3	.11+	.02+	.20+	.03+	.08+	.01	7+

* The sign + before or after a figure indicates that in this number of animals in each group there was a series or number of successive convulsions, *i. e.*, six of the seventeen control animals had a long series of successive convulsions.

that of the control group. This factor in some cases, as in intravenous injection of tapwater or of ammonium carbonate or in the case of removal of the adrenals, is zero which means that the dose necessary to produce the first convulsion, from which a control cat recovers, is in these cases sufficient to produce death. The table shows no marked variation in the average number of convulsions except in cats that have undergone anrenalectomy, in which the average is 1.4.

Table II shows that in all groups dilatation of the stomach is the most prominent gastro-intestinal symptom observed. It is present when the adrenals are removed. Dilatation of the colon is next in frequency, and contraction of the colon is next after dilatation.

The neurosurgical procedures are of importance in considering dilatation of the stomach. Cutting the splanchnics should make movements more distinct and increase the contractility or spasticity.[16] In these experiments we find the opposite to be true, *i. e.*, dilatation of the stomach and contraction of the pylorus in one case, while in another there was a spastic and contracted colon when the right splanchnic was cut. Bilateral section of the vagus nerve should have an opposite effect,[17] *i. e.*, no decrease of dilatation. In two cases in which both vagi were cut, moderate dilatation of the stomach was found in animals dying without injection of absinth. Dilated stomachs were noted on injection of absinth in animals after longitudinal section of the midbrain, transverse section of the cord at the level of the fifth or sixth thoracic and lower lumbar, segments. After ablation of both motor areas, a normal or slightly dilated stomach with spasm of the pylorus and contracted colon was noted in two cases. The type of convulsion, *i. e.*, tonic or clonic, does not seem to affect the gastro-intestinal reaction. A condition observed in almost every case is the fullness of the gall bladder, and contraction of the common bile duct to such an extent that even a great deal of pressure does not expel the contents. These observations seem to contradict the theory of gall bladder emptying as a result of differences in abdominal pressure due to respiratory movements,[18, 19] since these respiratory variations are rather marked in convulsions.

Table III indicates the almost even distribution of the size of the spleen and thymus in the above cases and presents data on the

TABLE II.

Type of experiment.	No. of cats.	Stomach.			Small intestine.			Colon.		
		Dil.	Control.	Normal.	Dil.	Control.	Normal.	Dil.	Control.	Normal.
Controls	17	8	1	8	0	P	16	2	1	14
Surgical procedure	16	12	0	4	0	3-P	12	3	2	11
Intravenous glucose	13	10	0	3	0	0	13	1	1	11
Intravenous tapwater	8	5	0	3	0	0	8	1	0	7
Removal of adrenals	13	5	0	8	0	0	13	0	0	13
Adrenalin	4	1	0	3	0	0	4	0	0	4
Nephrectomy	5	1	0	4	0	3	2	0	4	1
Ammonium carbonate	3	3	0	0	0	1	2	1	2	0
Luminal	6	5	0	1	0	P	5	0	0	6
Borotartrate	3	0	0	3	0	0	3	0	0	3

TABLE III.

Type of experiment.	No. of cats.	Thymus.			Spleen.			Circulation.			
		Large.	Small.	Normal.	Large.	Small.	Normal.	Lungs.	Kidneys.	Pancr.	Brain.
Control	17	6	4	7	4	3	10	8	3	0	0
Surgical procedure	16	4	5	7	2	4	10	10	4	1	1
Intravenous glucose	13	4	1	8	1	1	11	4	3	1	0
Intravenous tapwater	8	3	3	2	0	1	7	2	2	1	1
Removal of adrenals	13	4	3	6	2	1	10	7	2	0	1
Adrenalin	4	0	2	2	1	2	1	1	1		1
Nephrectomy	5	0	2	3	0	3	2	5	0	1	0
Ammonium carbonate	3	2	0	1	0	0	3	3	0	2	0
Luminal	6	2	3	1	0	3	3	6	0	1	0
Borotartrate	3	2	0	1	0	2	1	2	0	0	0

frequency of petechial hemorrhages and congestion in the lungs, kidneys, pancreas and brain occurring independently of or unaffected by the various experimental procedures. These might possibly explain some metabolic, pathologic and other findings mentioned so frequently in most of the papers on epilepsy.

CONCLUSIONS.

1. In animals after various surgical procedures upon the nervous system; intravenous injections of glucose, tapwater, adrenalin, ammonium carbonate, luminal and potassium borotartrate; as well as after adrenalectomy and nephrectomy, we find an approximation of the minimal convulsive to the lethal doses of absinth in experimentally produced convulsions as compared with the control animals.

2. When tapwater or ammonium carbonate is injected intravenously the "convulsive factor" is almost zero per cat and per pound as compared with 0.21 and 0.03 gram respectively in the control animals.

3. There seems to be a reduction in the number of convulsions after adrenalectomy.

4. Dilatation of the stomach is first in frequency, that of the colon next, and contraction of the colon last in the groups considered.

5. Some central and peripheral lesions of the nervous system show constant effects in dilatation or contraction of the parts of the digestive tube. These are not necessarily in accordance with accepted neurophysiologic principles.

6. Emptying of the gall bladder does not seem to be affected by the violent respiratory reactions met with in experimentally induced convulsions, since this organ is distended with bile and resists its expression in many instances.

7. The occurrence of extensive and widespread petechial hemorrhages in the lungs and congestion of other organs is a constant finding in all groups of experimentally produced convulsions considered above.

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DISCUSSION.

DR. F. H. PIKE (New York City).—This work represents just a cross section, without any attempt at analysis or explanation. There is a possibility that a great many of these gastro-intestinal conditions mentioned in the reports of autopsies on epileptics may have been the result rather than the cause. Dr. Hodskins' remark on the prevalence of a dilatation of the stomach brings that out. It is one of those things which we cannot explain at present. We simply want to know how closely our experimental measures parallel the clinical observations. We find the gastro-intestinal symptoms to be very constant. Sometimes they are alleviated by drugs, sometimes not.

DR. S. BERNARD WORTIS (New York City).—This is very interesting material, but we must not be over-enthusiastic in drawing conclusions. Those of us who have been on a general surgical service have seen dilatation of the stomach follow amputation of a leg or after general anesthesia for tonsillectomy. Stomach dilatation is not always dependent on a brain lesion, if ever! There is very often a dilatation of the stomach associated with our experimental convulsions in animals.

I think it is very important that these observations be presented. I also think we must take them with a grain of salt.

DR. S. M. WEINGROW (New York City).—As far as the remark by Dr. Wortis is concerned, that we must not emphasize the central or peripheral nervous control of the gastro-intestinal phenomena, I emphasized the point at the beginning of the paper that in most of the cases it is not so much that the control of the nervous system of the stomach produces dilatation, but that with the various procedures outlined we also got gastro-intestinal muscular phenomena.

Furthermore, Cannon has proved independent gastro-intestinal motility by the injection of croton oil into the cecum. He has also proved it by crushing various organs in the abdomen and producing a dilatation of the stomach. So dilatation of the stomach is definitely not a central phenomenon alone.

With regard to Dr. Hodskins' remark, I may mention that not only are functional gastro-intestinal phenomena observed at autopsy, but that a number of years ago—I think it was about 1911 or 1912—there was presented at one of the meetings of the American Association for the Study of Epilepsy a paper by A. E. Taft, who pointed out that in the course of convulsions the body organs presented specific infiltrations and other pathologic phenomena.

A STUDY OF THE EYE FINDINGS IN A GROUP OF EPILEPTICS, WITH REFERENCE TO THE MECHANICAL THEORY OF EPILEPSY.*

By HARRY COSTEFF, M. D.,
Peoria State Hospital, Peoria, Ill.

The question of the nature and origin of epilepsy has occupied the minds of scientific men from time immemorial. Numerous theories have been advanced but the ultimate etiology of epilepsy still remains the great unknown. Why a given condition will produce convulsive seizures in one individual and will fail to do so in another is a question that has never been answered. The mechanism of the convulsion, however, and the factors which help to produce it have been the subject of a great deal of study in late years, and we are probably nearer now than ever before to an understanding of the more important predisposing factors.

In the last few years the mechanical theory of epilepsy has been supported by Temple Fay [1, 2] and others. This theory has the merit of simplicity and throws considerable light on the various phenomena of epilepsy. According to Fay the most important underlying cause of epilepsy is an abnormal increase in intracranial pressure. This condition is brought about by the excessive accumulation of cerebro-spinal fluid over the cerebral cortex due to the faulty functioning of the sub-arachnoid villi, or the pacchionian bodies. It is the special function of these organs to filter the cerebro-spinal fluid and facilitate its drainage into the venous circulation. Hemorrhage into the sub-arachnoid space, especially that which occurs in the child during the course of difficult labor, inflammation, and trauma, are the most important causes for the subsequent impairment of the function of the pacchionian bodies.

To quote Temple Fay: [3]

> With sclerosis consequent to hemorrhage, inflammation or trauma, or impairment of the fluid spaces in the presence of a supracortical edema sufficient to stretch the thickened and adherent arachnoid attachments, there

* Due acknowledgment is made of the valuable help given by M. C. Whitlock, M. D., Ophthalmologist, of Peoria, Ill., in the diagnosis of the various eye conditions found in this series.

would be produced a form of subminimal stimulation that would be a predisposing, sensitizing generalized factor (at times focal in post-traumatic cases), variable depending upon fluid intake and overloading of the compensating threshold of the absorption mechanism (subarachnoid villi-pacchionian granules).

That in the presence of a circulatory disturbance (cerebral anemia) there would be produced increased intracranial pressure with further transudation of fluid into the tissues (anoxemia), increased arachnoid distension and traction due to arterial pulsation, these form subminimal stimuli, the summation of which precipitate or predispose to the final discharge of motor cortical impulses through the final common pathway for such movements.

In further elucidation of his theory Fay [4] states:

The factors surrounding the deficiency in cerebrospinal fluid elimination must be variable within a wide limit, so that certain cases would show almost complete compensation for overloads or retention of fluid, and other cases represent the poorest type of compensation for this fluid variable.

Starting with this hypothesis, and assuming that a long continued increase in intracranial pressure would be likely to manifest itself in various pathological conditions of the fundus of the eye, we undertook the present study.

The problem was formulated as follows:

1. Is there a sufficient number of epileptics whose eye-grounds show distinct pathology?

2. Are such conditions, if found, permanent or transitory in character?

3. May such pathological conditions be interpreted as proof of an existing increase in intracranial pressure?

A search through the literature did not reveal much material on this subject. The eyes of epileptics have been studied by Rodiet, Pansier, and Cans [5] who found the permanent disturbances to be: pallor of the optic disc and retina; passive venous congestion of the fundus with a pale disc; often intense blackish pigmentation of the choroid and retina. They also found optic neuritis and advanced gray atrophy in cases where the epilepsy was of long duration.

Penichet [6] reported fundus changes in nine cases of a total of 41 epileptics examined by him. The change noted was hyperemia of the disc during the first two or three days following the seizure. The retinal vessels behaved unsteadily, according to him.

Block [7] reported 100 cases of brain tumor, of which number 39 had convulsions. While choked disc occurred in only 39 out of 61

cases of patients who did not have any convulsions, it occurred in 31 out of 39 patients who did have convulsions.

Spratling [8] quotes Hughlings-Jackson who described retinal epilepsy, a condition of spasm in the retinal vessels during an epileptic attack which causes momentary blindness.

Our group consisted of 114 epileptics of whom 48 were males and 68 females. Their ages ranged from 17 to 77. The shortest duration of the disease recorded was four years. The greatest number had grand mal seizures for many years while some had petit mal as well as grand mal attacks. Those cases showing pathology were observed at different intervals over a period of six months and the changes noted. Many of them were observed shortly after attacks. We have not succeeded in examining any fundus during a seizure.

The most frequent condition found was passive hyperemia of the fundus. By passive hyperemia is meant a disturbed ratio between the retinal arteries and veins whereby the veins exceed their normal ratio to the arteries which is 3:2. In some cases the fundus was darker than normal, while in others it was not. The number of cases of passive hyperemia either with or without other pathology was 32. Out of this number 27 did not have any other pathology. In two cases this condition was associated with optic atrophy; in two others with papilitis; while in one case it was associated with retrobulbar neuritis. In 18 cases the disturbed ratio persisted during the whole period of observation, while in 14 cases it cleared up at times during the interparoxysmal period.

Next in frequency was optic atrophy. There were 17 cases in this group. In 10 there was no other pathology. In three cases the condition was associated with choroiditis, while in three other cases it was associated with passive hyperemia. In one case it was associated with retrobulbar neuritis.

Choroiditis either alone or with other conditions was found in 11 cases. In seven there was no associated pathology. In three cases it was associated with optic atrophy, while in one case it was associated with coloboma of the optic nerve.

There was one case of iritis, one case of papilitis associated with passive hyperemia, and two cases of retrobulbar neuritis.

Coloboma of the optic nerve was found either alone or associated with choroiditis in two cases.

In nine cases the fundus could not be seen, either due to cataracts, lack of cooperation of patient, or other causes.

In 46 cases the fundus was normal.

In summarizing our results we find that there was distinct pathology in 58 out of 114 cases. The number would probably have been greater had we been able to examine the nine doubtful cases. Thus the first question, whether there is a considerable number of epileptics showing pathology in the fundus, was answered in the affirmative. Over 50 per cent of our cases showed distinct pathology in the fundus.

In answer to the second question our findings show that only in 14 of the 58 cases where pathology was found was the condition transitory in character. In the great majority of cases the condition proved to be permanent.

For an answer to the third question, whether in our study we found any proof of increased intracranial pressure, we would have to look to the passive hyperemia group. A disturbed ratio in the blood vessels of the fundus could logically be assumed to be caused only by some interference with the return circulation of the fundus of the eye. In the absence of any systemic cause, as general hypertension, it would likely be caused by an increase in intracranial pressure. Local conditions in the eye itself which might cause the passive hyperemia were carefully excluded. The blood pressure taken on the group of passive hyperemia cases was if anything below normal. Several patients registered a systolic pressure below 100 and a diastolic pressure between 60 and 70. In one case the diastolic pressure was as low as 55 and the systolic 85.

The close relationship between intracranial pressure and the pressure in the retinal vessels has been shown by Kalt.[9] He measured the pressure in the central artery of the retina by means of Bailliart's ophthalmodynamometer in a series of cases. He found a parallelism between the intracranial pressure and the pressure in the retinal arteries in almost all cases. He found that the pressure in the retinal arteries faithfully follows all the variations of the cerebro-spinal pressure; it increases immediately when one compresses the brain of a trephined person; and it decreases when there is a subarachnoid decompression by a lumbar puncture or injection of hypertonic solutions. Bailliart came to the conclusion that there is a permanent equilibrium between the venous pressure and the

pressure of the surrounding medium—in this case the cerebro-spinal fluid. Under pathological conditions this equilibrium may be disturbed. When there is increased pressure of the cerebro-spinal fluid there is obstruction to the circulation of the veins and capillaries, and there may be a passive compensatory increase in the arterial pressure. This would show itself in the raised pressure of the retinal artery, especially in the diastolic pressure, which he considers by far the most important. It is understood that general circulatory hypertension must be excluded. Claude, Lamache and Dubar,[10] in a similar work came to the same conclusion. They found a low retinal pressure to indicate intracranial hypotension; and retinal hypertension, in the absence of general hypertension, to indicate intracranial hypertension. In a subsequent paper these authors [11] corroborated their previous findings.

The main point of interest to us is the unanimous conclusion at which these workers arrive that the diastolic pressure in the retinal artery faithfully registers the degree of intracranial pressure. The diastolic pressure anywhere in the body is determined by the amount of blood in the veins at any particular moment. The diastolic pressure in the retinal artery would then be determined by the amount of blood in the retinal veins. Venous engorgement of the fundus would therefore be synonymous with an increase in the diastolic pressure of the retinal artery, since the former would be immediately responsible for the latter. Since a rise in the diastolic pressure of the retinal artery indicates almost invariably a rise in intracranial pressure whenever there are no other conditions to account for it, we may logically assume that venous engorgement of the fundus, or passive hyperemia, would indicate the same condition.

In our group 32 cases showed passive hyperemia. It constituted 55.1 per cent of the total pathology found, and 30 per cent of all the cases in which the fundus was actually examined. The fact that such a large group showed identical pathology is significant. It tends to show that at least in a considerable number of epileptics there is distinct evidence of increased intracranial pressure. The intracranial pressure may not always be of the same degree. Fay [12] explains this circumstance by the fact that "the factors surrounding the deficiency in the spinal fluid elimination must be variable within a wide limit." This capacity probably varies also in the same person at different times, which would explain the occasional clearing up of the passive hyperemia in 14 of our cases.

We feel that in so far as the group of passive hyperemia cases is concerned, we are fairly safe in interpreting the fundus pathology as signifying the existence of intracranial hypertension.

CONCLUSIONS.

The fundi of 114 epileptics were studied with the object of ascertaining the presence or absence of increased intracranial pressure. Of this number 58 cases showed distinct pathology while nine could not be examined. The most frequent pathology found was a venous engorgement of the eye fundus. This group constituted 55.1 per cent of the total pathology found. Of these 32 cases of passive hyperemia 18 persisted while 14 occasionally cleared up during the interparoxysmal period. Temple Fay's mechanical theory of epilepsy is reviewed and discussed in relation to our findings. The work of Kalt, Claude, and others on the relation between intracranial pressure and the pressure in the retinal vessels, is also referred to. The conclusion is reached that the relatively frequent incidence of passive hyperemia in the fundus of epileptics would tend to prove the existence of intracranial hypertension in at least a considerable number of cases.

COMMENTS.

Over 50 per cent of our series showed pathology of the fundus. This greatly exceeds the findings of Penichet who found pathology in somewhat over 24 per cent. We did not always find as he did, a hyperemia of the disc during the first few days following the seizure. We often found only a venous distension with a clear fundus. In two or three cases we did not find a disturbed ratio following the seizure. Those of course were exceptional. As a general rule wherever there is a disturbance in the ratio it is more marked following the seizure. It persists for a variable period, clearing up in some cases but apparently remaining permanent in others.

We have found a considerable number of cases of optic atrophy, especially in the old deteriorated cases. This would agree with the findings of Rodiet, Panser and Cans.

While the present study was undertaken primarily with reference to the mechanical theory of epilepsy, we believe that the findings are quite interesting in themselve and deserve further study.

TABLE SHOWING SEX, AGE, TYPE OF SEIZURE AND EYE PATHOLOGY IN ALL EPILEPTICS EXAMINED, ALSO BLOOD PRESSURE IN THE PASSIVE HYPEREMIA GROUP.

	Sex.	Age.	Kind of seizure.	Eye pathology.		Blood pressure.
1.	F	?	G. M.	Passive hyperemia		?
2.	M	53	"	"	"	120/70
3.	M	35	"	"	"	120/90
4.	M	29	"	"	"	110/80
5.	M	46	"	"	"	110/80
6.	M	26	"	"	"	120/90
7.	M	46	"	"	"	138/80
8.	M	47	"	"	"	130/90
9.	M	62	"	"	"	110/80
10.	F	29	G. M. and P. M.	"	"	95/50
11.	F	30	G. M.	"	"	130/100
12.	F	44	"	"	"	120/80
13.	F	53	"	"	"	120/90
14.	F	37	"	"	"	120/80
15.	F	27	"	"	"	120/70
16.	F	46	"	"	"	120/80
17.	M	56	"	"	"	130/90
18.	F	44	"	"	"	110/80
19.	M	56	"	"	"	120/80
20.	F	49	"	"	"	88/56
21.	F	38	G. M. and P. M.	"	"	130/80
22.	M	35	G. M.	"	"	130/100
23.	F	33	"	"	"	130/90
24.	M	77	"	"	"	120/90
25.	F	17	"	"	"	90/60
26.	M	43	"	"	"	120/90
27.	M	51	"	"	"	120/80
28.	M	29	"	Passive hyperemia c. low grade choroiditis		?
29.	F	55	"	Optic atrophy c. passive hyperemia		120/80
30.	F	49	"	Retrobulbar neuritis O. D. c. passive hyperemia O. U.		?
31.	F	35	"	Papilitis c. passive hyperemia		110/70
32.	M	64	"	Optic atrophy O. S. c. passive hyperemia O. U.		130/90

TABLE SHOWING SEX, AGE, TYPE OF SEIZURE AND EYE PATHOLOGY IN ALL EPILEPTICS EXAMINED, ALSO BLOOD PRESSURE IN THE PASSIVE HYPEREMIA GROUP. —CONTINUED.

	Sex.	Age.	Kind of seizure.	Eye pathology.	Blood pressure.
33.	M	65	G. M.	Optic atrophy c. active choroiditis	
34.	F	35	"	Optic atrophy c. choroiditis O. S.	
35.	F	65	"	Optic atrophy c. choroiditis	
36.	F	70	"	Optic atrophy c. retrobulbar neuritis	
37.	M	56	"	Optic atrophy O. D.	
38.	F	30	"	Optic atrophy O. S.	
39.	F	?	"	Optic atrophy O. D.	
40.	F	39	"	" "	
41.	M	57	"	Optic atrophy	
42.	F	57	"	Optic atrophy O. D.	
43.	M	49	"	Optic atrophy	
44.	F	52	G. M. and P. M.	" "	
45.	F	37	G. M.	" "	
46.	M	37	"	" "	
47.	F	53	"	Choroiditis	
48.	M	38	"	"	
49.	M	49	"	"	
50.	F	55	"	Low grade choroiditis	
51.	M	56	"	Choroiditis O. S.	
52.	F	67	P. M.	Choroiditis (chronic)	
53.	F	60	"	Choroiditis c. coloboma of optic nerve	
54.	F	64	G. M.	Choroiditis	
55.	F	42	"	Iritis O. D.	
56.	F	67	"	Retrobulbar neuritis (chronic)	
57.	F	62	"	" " "	
58.	M	47	"	Coloboma of optic nerve O. S.	
59 to 105 incl.—Age range 27-76—				Fundus normal	
106 to 114	"			Fundus could not be seen	

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12. Fay, Temple: Convulsive Seizures. Am. Jour. Psych., January, 1931, p. 555.

CATATONIA IN ANIMALS.

Experimental Studies of the Effect of Bulbocapnine and Other Drugs.*

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This communication ventures to call attention to some possible toxic factors in the genesis of catatonia as suggested by experimental studies of the effects of bulbocapnine injections in animals. In his fifth edition of the Textbook on Psychiatry, Kraepelin regarded catatonia as a disorder of metabolism and this toxic conception of catatonia has been shared by a number of other well known clinicians such as Jelgersma, Régis, Sérieux and Claude. It may still be true that the best investigators have little to say regarding the toxic factors in schizophrenia[1] and no attempt will be made to suggest that toxic agents are the sole factors in the etiology of catatonia.

The study of experimental catatonia in animals induced by bulbocapnine injections had its origin in some plethysmographic studies by de Jong[2] in Amsterdam. In catatonic patients he found comparatively little response in the pulse tracings to the usual stimuli and concluded that this vascular rigidity was indicative of an organic and probably a toxic condition. It then occurred to him that he might be able to reproduce in animals this kind of pathological response in the vegetative nervous system and he began a

* Read at the eighty-seventh annual meeting of The American Psychiatric Association, Toronto, Canada, June 1-5, 1931. This work was made possible by the Bloomingdale Hospital Fellowship in Psychiatry.

[1] Meyer, A.: The Nature and Conception of Dementia Præcox, J. Abn. Psychol., 5: 274.

[2] de Jong, H.: Die Hauptgezetze einiger wichtigen Körperlichen Erscheinungen beim psychischen Geschehen von Normalen und Geisteskranken, Ztschr. f. d. ges. Neurol. u. Psychiatr., 1921.

search for some toxic substance by which he might induce an artificial catatonic state.

Among the drugs suggested to him for this purpose was bulbocapnine, a drug whose remarkable properties are comparatively unknown by the medical profession. It is an alkaloid resembling apomorphine obtained from the plant corydalis cava which in the middle ages was known for its medicinal properties.

In 1904, Peters, a pharmacologist, observed that when he injected into animals certain doses of bulbocapnine (20 mg. per kg. wt. of animal) the animals became immobilized and although they appeared to be aware of the stimuli, they failed to manifest the usual motor response. He noticed that these animals were not paralyzed and that they were even capable of standing on three feet.[3] No attempt was made to compare this artificial catatonic state in animals with the human catatonia until de Jong began his experiments in 1921. From time to time other students of this aspect of catatonic conditions have collaborated with de Jong. This is particularly true of Baruk who with Professor Claude in the psychiatric clinic of the University of Paris had been conducting physiological and neurological studies of the catatonic motor syndrome.[4] The work of de Jong and Baruk has already been reported in their numerous publications[5] and my studies merely corroborate and supplement theirs.

[3] Peters, F.: Pharmakologische Untersuchungen über corydalis alkaloide, Archiv f. exp. Pharmakol. u. Pathologie, 1904, Bd. 51, pp. 130-173.

[4] Claude, H., and Baruk, H.: La catatonie. Étude clinique et physiologie pathologique, La Presse Médicale, No. 103, décembre, 1928.

Baruk, H.: Algunas Consideraciones Nuevas sobre las Afecciones Organicas y Organo—psiquicas cerebrales, Revista argentina de Neurologia, Psichiatría de Medicina legal, 1928.

[5] A general presentation of these bulbocapnine experiments will be found in a book by de Jong and Baruk entitled "La catatonie expérimentale par la balbocapnine" published in Paris by Masson, 1930. Further details may be obtained from the following publications of these authors:

Étude comparative expérimentale et clinique des manifestations du syndrome catatonique. Soc. de Neurol. de Paris et Revue Neurologique, janvier, 1929, p. 21.

Études sur la catatonie expérimentale. L'épreuve de la bulbocapnine chez la grenouille et la souris. Proceedings Académie des Sciences d'Amsterdam, Vol. XXXII, no. 7, 1929, p. 940.

In these reports attention has been called to the great similarity between the catatonic state artificially induced by injections of this drug and that observed in human beings. de Jong and Baruk have observed that such phenomena as the loss of motor initiative, negativism and catalepsy can be readily induced by suitable doses of bulbocapnine in the most highly developed animals, particularly the mouse, the cat and the monkey, whereas in animals such as the frog and the fish these phenomena are entirely lacking. It appeared from this that the production of these hypokinetic phenomena was dependent upon the presence of the neo-cortex. If this is true, it might be assumed that those animals having a rudimentary neo-cortex would present some of the catatonic phenomena.[6]

It was my privilege while working in the laboratory of de Jong and in the neurological clinic of Professor Brouwer to perform some experiments which were calculated to demonstrate the extent to which this assumption might be true. For this purpose were selected several different kinds of birds[7] all of whom have an equivalent development of the neo-cortex with the exception of the parokeet in which it is somewhat more highly developed.

Études sur la catatonie expérimentale. L'épreuve de la bulbocapnine chez la poule. Catalepsie et sommeil. Proceedings Académie des Sciences d'Amsterdam. Vol. XXXII, no. 7, 1929, p. 947.

L'épreuve de la bulbocapnine chez les singes. Comparaison des stades de l'intoxication avec les aspects de la catatonie humaine. Soc. de Neurol., Paris, 7 nov., 1929. Revue Neurol., nov., 1929, p. 54.

L'épreuve de la bulbocapnine chez des animaux avec et sans neopallium. Soc. de Neurol., 7 nov., 1929, R. N., nov., 1929, p. 532.

La catatonie expérimentale par la bulbocapnine et le syndrome catatonique chez l'homme. Étude comparative, L'Encéphale, fevrier, 1930, p. 97.

Pathogénie du syndrome catatonique et catatonie expérimentale. L'Encéphale, mars, 1930, p. 180.

[6] Ariens Kappers, C. U. Vergleichende Anatomie des Nervensystems der Wirbeltiere und des Menschen, Haarlem, 1920.

Kappers distinguishes in the development of the cortex two older parts, the archi-cortex and the palæo-cortex, and a newer part, the neo-cortex. The largest part of the cerebral cortex in man is neo-cortex. A beginning of the neo-cortex is found in birds ("Sagittalwulst") while in the lower animals none is present. For further details regarding the cortex of birds see Dennler, G., Folia Neuro-Biologica, 1922, No. 2, p. 343.

[7] In these experiments the finch (fringilla coelebs), the pigeon (columba domestica), the parokeet (melopsittacus parundulatus), the siskin (crysomitris spinus), the hen (gallus domesticus) and the duck (anas domestica) were used.

Differences in reaction were therefore dependent upon the amount of drug given to the animal.

The results of these experiments with birds have already been reported in some detail [8] and only a few of them will be included with the reports of experiments upon other animals so that a comparison may be made of the reactions to bulbocapnine injections according to the degree of development in the scale of evolution.

Previous experiments have demonstrated conclusively that the appearance of catatonic phenomena is dependent upon and varies according to the degree of bulbocapnine intoxication. This is especially true in the more highly developed animals. On this account the first injections into any given animal were the minimal for the production of manifestations of intoxication and the subsequent doses were gradually increased until many of the animals died from the immediate effects of the drug. There was always sufficient interval between experiments so that the animal might fully recover from the previous injections.

Only a few of the more illustrative experiments will be reported in detail and in addition note will be made of such reactions as will tend to complete the record of the findings.

LAND TURTLE NO. 1. WEIGHT 350 GM.

EXPERIMENT NO. 1.

10:15	A. M.	Injected 20 mgm. commercial bulbocapnine in thigh. Animal took a few steps and then remained motionless with leg slightly contracted.
10:18	"	Somewhat more restless—moving about slowly.
10:20	"	Walking, legs fully extended.
10:22	"	Quiet again but responds to stimuli.
10:30	"	No movement unless stimulated.
11:30	"	No change.
1:30	P. M.	No change.

EXPERIMENT NO. 2.

12:38	P. M.	Injected 40 mgm. commercial bulbocapnine.
12:40	"	Sitting with head fully extended—occasionally moving—walking slowly.

[8] Henry, G. W.: Catatonia in Birds, Psychiat. Quart., Vol. 5, pp. 68-81. Experimental Catatonia Induced by Bulocapnine in Birds, Psychiatrische en Neurologische Bladen, Jahrgang, 1930, No. 5.

12:42	P. M.	Walking more quickly.
12:43	"	Copious watery defecation.
12:44	"	Swallowing. Watery defecation.
12:45	"	Becomes quiet again.
12:48	"	Somewhat restless—keeps head fully extended.
12:50	"	Head withdrawn.
12:56	"	No change in responses to stimuli. Practically no spontaneous activity.
1:30	"	Sits with head withdrawn. Extends it when shell is tapped.

EXPERIMENT NO. 3.

10:23	A. M.	Injected 60 mgm. commercial bulbocapnine.
10:25	"	Restless movements of front legs and head.
10:26	"	Yawning and extending head.
10:30	"	Occasionally head and front legs are extended and withdrawn slightly in rhythmic manner.
10:35	"	Head fully withdrawn and then extended.
10:55	"	Same.
11:05	"	Seems hyperexcitable when stimulated. Inclined to extend head and legs when shell is tapped.

EXPERIMENT NO. 4.

11:58	A. M.	Injected 100 mgm. commercial bulbocapnine.
2:00	P. M.	Quite inactive—head withdrawn—practically no response to stimuli.
5:00	"	Same except that there are slow movements of the legs when the position is changed and further withdrawal of the head when the shell is tapped.
8:00	A. M.	(Next day.) Found dead.

The results obtained from the experiments upon the turtle are essentially in agreement with those of de Jong and Baruk who experimented in like manner upon fishes, frogs, lizards and salamanders. Small and moderate doses seem to have no effect and the large doses produced hyperkinetic phenomena which sometimes terminated in convulsions and death.

These animals are devoid of a neo-cortex and it will be interesting now to observe the responses to small and medium doses of bulbocapnine when injected into animals having a rudimentary neo-cortex. This will be shown in the following experiments with birds:

FINCH NO. 1 (FRINGILLA COELEBS). WEIGHT 16 GM.

(Bird easily frightened, very active, jumping about trying to escape from presence of human beings.)

EXPERIMENT NO. 1.

11:34	A. M.	Injected 1 mgm. bulbocapnine hydrochlorate.
11:35	"	Standing motionless on perch.
11:36	"	Cage stroked—does not leave perch. Apparently drowsy.
11:38	"	Still drowsy—opening and closing eyes slowly—yawning—little spontaneous movement.
11:39	"	Stimulated by loud noise and striking side of cage—bird jumps from perch to floor of cage and remains there. Moves about actively when series of stimuli given.
11:45	"	Left alone—becomes inactive again, apparently drowsy.
11:50	"	Becoming more active—jumping and flying from one perch to another—drinking a little water.
11:55	"	Without stimuli—again rather inactive—resting on perch.
12:00	M.	Put in dark room—a few minutes motionless—practically no attempt to withdraw from stimuli ordinarily provoking fright and attempts to escape. Easily aroused by being moved.
12:15	P. M.	In light room again—much more alert—eating, moving about—not so easily frightened as normally.
12:30	"	Apparently in normal state again.
3:00	"	Normal behavior in daylight. Taken to dark room—quickly becomes quiet—sitting on perch—not disturbed by stroking bars of cage, whistling, clapping hands except for momentary start at loud noises. When moved or touched withdraws slightly from stimulus chiefly to maintain equilibrium. Different degrees of light tried—bird is increasingly responsive and spontaneous activity is greater as room becomes light. Response to stimuli and spontaneous activity are reduced to minimum in darkness, greater in red light, still greater in yellow light and greatest in daylight.

EXPERIMENT NO. 2.

10:29	A. M.	Injected 1 mgm. bulbocapnine.
10:32	"	Resting quietly on bottom of cage.
10:34	"	Slightly drowsy—eyes half closed.
10:35	"	Drowsy, motionless. Does not react to stroking cage. Tries to fly when cage moved. Soon becomes quiet again.
10:38	"	Dark room—not responsive except to maintain equilibrium.
10:40	"	Light room—without stimuli except conversation and presence of observers—closes eyes and remains motionless.
11:00	"	Still quiet but flies away from approaching hand.

11:05 A. M. Injected ½ mgm. bulbocapnine—bird gasping occasionally within few seconds after injection.
11:07 " Motionless on bottom of cage except for swallowing movements.
11:08 " Shaking head.
11:10 " Shaking self and head—gasping.
11:12 " Unsteady on feet—nearly fell over—eyes closing—still gasping—slight tremor of wings.
11:14 " Settling down as though profoundly intoxicated. Still opening and closing beak slightly.
11:16 " Does not move from approaching hand. Head bent backward—slowly resumes usual position.
11:18 " Always maintains equilibrium. Put on perch—remains there. Aroused by being pushed.
11:20 " Taken out of cage—rests upon edge of saucer—makes no attempt to move.
11:24 " When pushed along, moves *en bloc*—feet sliding along on table. At same time can move legs. When put on edge of saucer resists being pushed off—even when one leg forced off clings with other and returns to perching position.
11:25 " Defecated. Saucer tilted—tries to maintain equilibrium until feet slide—then flies to desk and becomes motionless again. Always resists change in position.
11:40 " Taken to dark room in cage—put on perch—soon closes eyes—nods—remains motionless with eyes closed even when carried into light room provided equilibrium not disturbed.
12:03 P. M. No change. Beak placed in water—shakes beak and head violently, possibly slight swallowing movements but no active drinking. Goes to "sleep" again perched on edge of water bowl.

EXPERIMENT NO. 4.

10:20 A. M. Injected 3 mgms. bulbocapnine.
10:21 " Gasping. Defecated.
10:22 " Gasping. Shaking head as though trying to dislodge something from beak. Otherwise motionless, sitting on table, out of cage.
10:25 " Still gasping.
10:30 " Gasping more rapidly and feebly. Getting drowsy. Eyes closing.
10:33 " Sitting quietly, breathing rapidly, eyes closed, head to one side.
10:35 " Aroused slightly by pushing—opens eyes, takes few steps—resists being pushed—slides *en bloc* along top of table with feet braced.

10:38 A. M. Watery defecation. Occasional shivering movements. When pushed backwards resists *en bloc* and then takes few steps *forward*. When pushed ahead resists and then takes few steps *forward*. No spontaneous movements.

10:50 " Two more mgm. bulbocapnine injected.

10:52 " Watery defecation.

10:55 " Wings quivering. Difficulty maintaining equilibrium—rests with toes curled under in awkward position.

11:00 " Moves *en bloc* without taking step forward. When toes are crossed, maintains this awkward position. Watery defecation. Breathing 120 per min.

11:08 " Wings quivering. Drowsy—remains in sitting position. Beak put in water—merely shakes water off.

11:10 " Watery defecation.

11:13 " Head bent forward with stick—does not try to free itself—stick removed—head slowly resumes usual position.

11:15 " Wing raised with stick—tries to free itself but takes no steps aside or forward and unless wing greatly extended will tolerate unusual position.

11:22 " Pushed ahead *en bloc*. If continued will take few steps forward and then stop.

11:25 " Still permits head to be bent forward—slowly resuming usual position.

11:28 " Permits head to be held hyperextended and toes to be crossed—as long as equilibrium is not disturbed.

11:40 " Respirations 100 per minute. Sitting quietly with eyes half closed.

11:45 " No longer permits head to be held hyperextended or hyperflexed. Toes can still be held curled under. Bird pecks at stick in self-defense.

11:52 " Sat seven minutes with toes of both feet crossed.

11:55 " Easily aroused. Apparently annoyed by having beak moved by stick and pecks at it. Makes no spontaneous movements but when pushed takes few steps forward or aside according to direction of push.

12:00 M. No longer moves *en bloc* when pushed. Pushed forward—takes few steps and makes feeble attempt to escape. Put back in cage. Rests on perch—apparently still slightly drowsy when not stimulated.

12:10 P. M. Still sits dozing on perch.

2:30 " Sitting quietly on perch—not disturbed by approach of observer. Watery defecation.

3:15 " More active—jumping about from one perch to another but apparently not frightened and does not make the desperate attempts to escape, such as flying at side of cage, characteristic of its normal condition. Eats a few seeds.

4:30 P. M. Somewhat more active but not frightened by approach of observer—frightened slightly by striking side of cage with stick. Soon becomes composed and begins eating—drinks a little water and chirps.

PAROKEET NO. 1 (MELOPSITTACUS PARUNDULATUS). WEIGHT 27 GM.

EXPERIMENT NO. 10.

10:10 A. M. Injected 2 mgm. bulbocapnine hydrochlorate. Replaced in cage—rather excited at first—running on floor of cage—jumping on perch and down again. Apparently not unsteady.
10:13 " Becoming more quiet—sitting on floor of cage.
10:15 " Feathers of wings being erected slightly.
10:16 " Less active. Vomiting movements, slightly productive.
10:19 " Breathing much more deeply and more rapidly. (100 per min.)
10:23 " Sitting on floor of cage, somewhat crouched. Eyelids closing. Tries to escape when hand approaches. Apparently equilibrium slightly disturbed.
10:25 " Watery defecation. Sitting on perch. Eyelids closing occasionally.
10:26 " Definitely ataxic in movements. Swaying slightly backwards and forwards. Sits on floor of cage with eyes closed part of time.
10:33 " Removed from cage. Bites hand of observer. Flies about 3 feet—then remains quiet. Seems to prefer dark corner.
10:35 " Sits on table in front of observer. Dozing, making no attempt to move, eyes closed most of time.
10:39 " Allows beak to be raised and head hyperextended by stick for 1 minute but when startled flies away.
10:40 " Allows feet to be placed in awkward positions but slowly returns them to near normal position.
10:44 " Permits head to be forced to top of table and held there—slowly raises it again.
10:45 " Rather easily aroused by being pushed. Watery defecation. When pushed takes a few steps.
10:55 " When approached with stick flies half way across room. Put back in cage. Sits on floor with eyes closed. Opens eyes when wires of cage are stroked.
12:15 P. M. Less active than normally. Not so easily startled and does not attempt to escape. Sits on perch.
3:35 " Apparently has regained usual condition.

HEN NO. 1 (GALLUS DOMESTICUS). WEIGHT 1500 GM.

EXPERIMENT NO. 14.

2:40 P. M. Injected 60 mgm. commercial bulbocapnine.
2:42 " Swallowing movements—walking about slowly—breathes with mouth open.

2:44 P. M. Breathing deeply and rapidly. Swallowing. Apparently very alert. Stands in one position.

2:47 " Allows feet to be pushed short distance—then walks away—cackles a little—remains near observer—breathing deeply.

2:49 " Watery defecation. Allows body to be pressed to floor, then takes a few steps away and becomes stationary again.

2:53 " Withdraws slightly from threatened blows. Breathing deeply and rapidly with mouth open.

2:55 " Same. Resists slightly being pushed to either side—takes few steps and then remains fixed.

2:58 " Permits itself to be picked up (when done gently) and moved from one part of floor to another. Remains standing with toes curled under.

3:03 " Remains standing with feet spread apart and one raised 1 inch above the other.

3:05 " Remains standing with one leg 4 inches above the other and feet spread apart. Watery defecation.

3:08 " Settling down. Closes eyes temporarily. Breathing more rapidly.

3:10 " Permits head to be bent to floor and held there, then slowly resumes usual posture. Stands with legs spread apart.

3:15 " Rather easily aroused—runs away from stick—flaps wings, cackles—then immediately resumes motionless state. Eyes closing at times.

3:20 " Placed on side—remains there. Rolled gently over on back—remains thus.

3:23 " Placed on perch—squats—with wings spread widely. Breathing very rapidly and forcefully with mouth open.

3:25 " Both wings pulled out—resting on edge of box—remains thus. Defecation. Sits with eyes closed part of time. No spontaneous movements.

3:30 " Forced from perch—jumps to floor and then remains motionless.

3:35 " One leg raised 4 inches put on top of box and widely separated from other—settles down to squatting position. Two minutes later assumes more comfortable and normal posture.

3:40 " Put in basin—stands in water and allows water to run continuously on its back. Offered water—makes no attempt to drink.

3:45 " Standing half immersed in water without making any effort to move. Forced to fly 3 feet to floor—makes no further effort to move.

3:50 " When undisturbed, sits motionless, eyes closed, one wing extended.

4:00 " Same. Comb has very deep red color.

4:10 " Sits breathing with mouth open after being forced to fly. Then closes eyes and remains motionless.

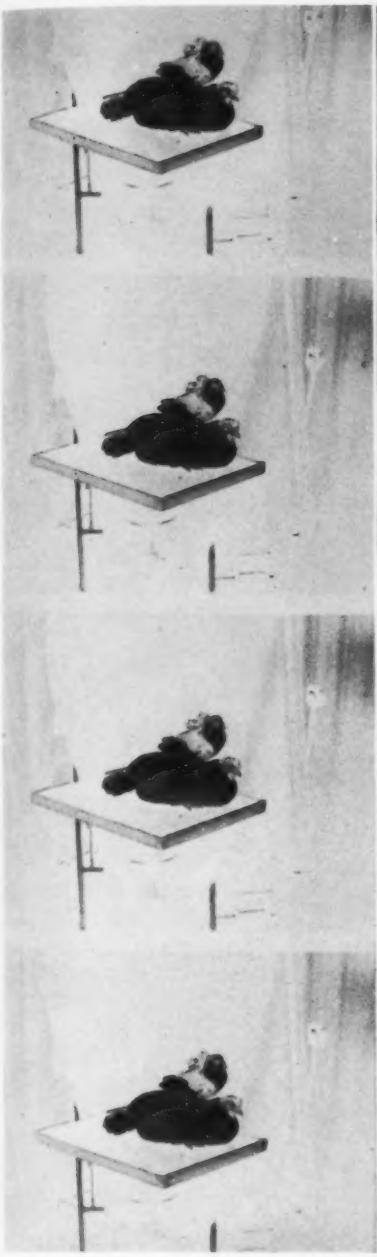

FIG. 1.—Catatonic hen, pigeon and finch remain undisturbed although four observers are moving about and talking nearby.

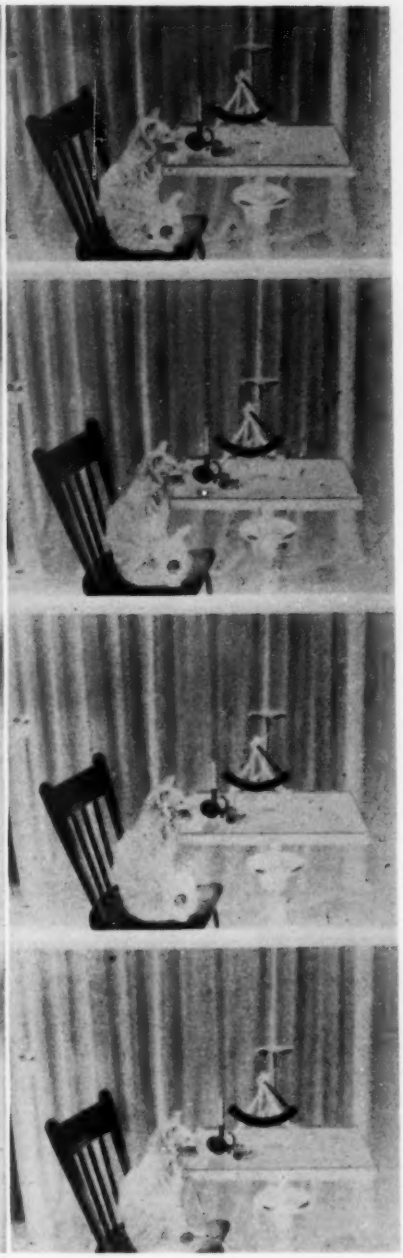

FIG. 2.—Catatonic cat sits in unusual position within a few inches of a catatonic mouse and bird. No indication of the usual hostile relationship was observed.

SECTIONS OF MOVING PICTURE FILM SHOWING ANIMALS IN CATATONIC STATE FOLLOWING INJECTIONS OF BULBOCAPNINE.

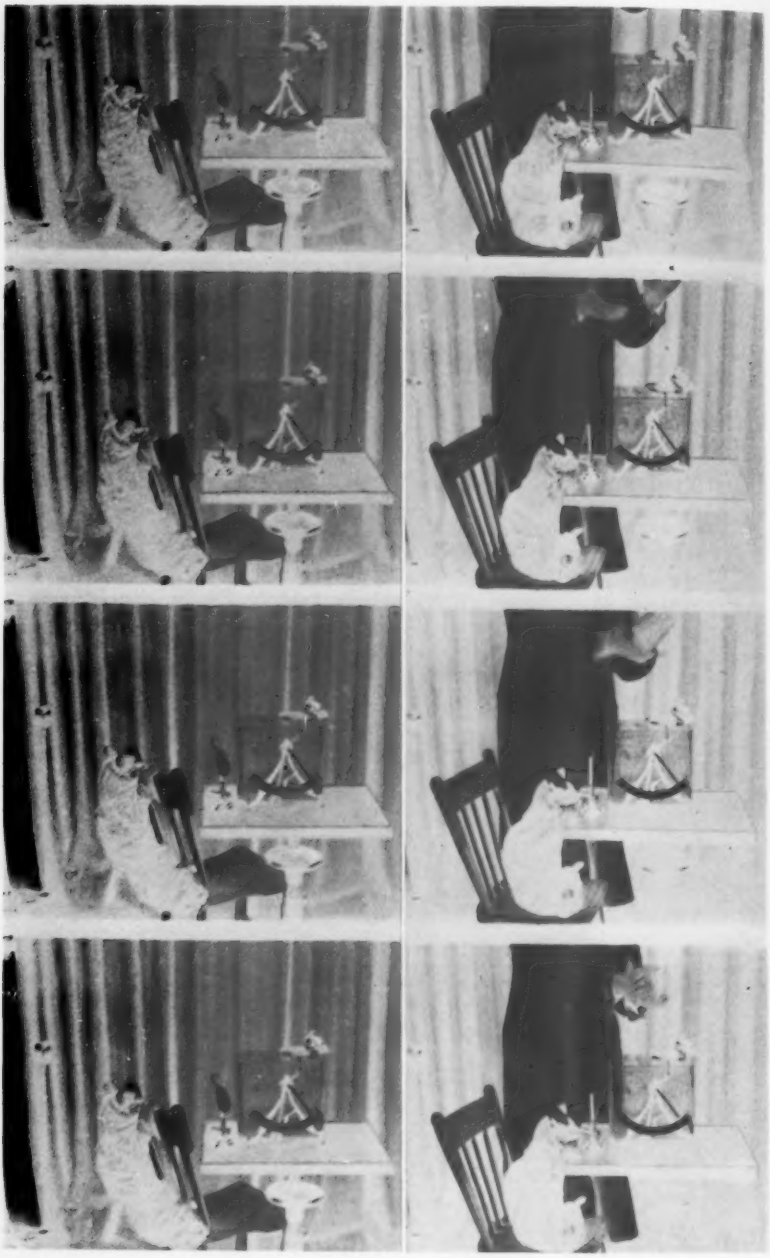

FIG. 3.—Catatonic cat, mouse and bird. The cat hangs for an indefinite period from the back of a chair. The bird remains immobile on a delicate letter weighing balance upon which it is impossible to place a normal bird.

FIG. 4.—Catatonic cat, mouse and bird. The mouse remains immobile on the face of the cat, and the bird on the platform of the balance in spite of the fact that an observer is clapping his hands loudly in their presence.

SECTIONS OF MOVING PICTURE FILM SHOWING ANIMALS IN CATATONIC STATE FOLLOWING INJECTIONS OF BULBOCAPNINE.

PIGEON NO. 3 (COLUMBA DOMESTICA). WEIGHT 320 GM.

EXPERIMENT NO. 23.

11:20	A. M.	Injected 3½ mgm. pure bulbocapnine.
11:22	"	Swaying slightly from side to side.
11:24	"	Defecated. Vomiting movements.
11:30	"	No spontaneous activity.
11:45	"	Same. Out of cage—flies short distance when stimulated—then remains motionless.
12:15	P. M.	Same. Injected 3 mgm. pure bulbocapnine.
12:20	"	Placed on table to be photographed—remains motionless.
12:22	"	Stimulated with stick—flies to ground.
12:30	"	Placed on injected hen's back and remains motionless.
12:31	"	Injected finch placed on back of pigeon while still on hen's back. All three birds remain inactive.
12:32	"	Finch stimulated with stick—flies away.
12:33	"	Pigeon stimulated—flies to floor.
1:00	"	Sits motionless in cage.
2:00	"	Somewhat more alert. Moves away from approaching hand—then motionless.
2:30	"	Sits dozing.
3:30	"	More active.
4:30	"	Apparently normal again.

HEN NO. 1.

EXPERIMENT NO. 24.

11:25	A. M.	Injected 60 mgm. pure bulbocapnine.
11:28	"	Breathing rapidly with mouth open. No spontaneous activity.
11:29	"	Defecated.
11:40	"	Same. No spontaneous activity. Still breathing rapidly, with mouth open at intervals.
12:00	M.	Permits one leg to be supported 5 inches above the other.
12:15	P. M.	Has remained in this awkward position. Dozing at times.
12:20	"	Photographed with leg still raised.
12:25	"	Stimulated with stick—flies about, coming to rest again when not stimulated.
12:30	"	Injected pigeon and finch placed on hen's back—all remain motionless.
12:33	"	Finch and pigeon both fly away after being stimulated. Hen remains motionless.
12:34	"	Stimulated with stick—flies away but immediately comes to rest on cessation of stimulus.
1:00	"	Sits dozing.
2:00	"	Much more alert, moving about slowly, eating.
3:00	"	Dozing at times. At other times alert and active.
4:30	"	Apparently normal again.

The experiments with birds are reported somewhat more fully because with the exception of a few observations on pigeons [9] there has been no previous study of the reactions of birds and they permit the following general conclusions to be drawn.

Cessation of bodily movements and drowsiness appear soon after bulbocapnine injection. The behavior observed is similar to that of the normal bird when placed in a dark room. The condition is different from that of sleep, however, since it occurs in bright daylight, without the preliminary groping about shown by the normal bird in darkness before settling down. When a bird is only mildly intoxicated it responds to stimuli by making attempts to escape whereas the sleeping bird does little but maintain equilibrium. The normal bird gradually resumes its usual activities with increasing illumination while the behavior of the injected bird remains essentially unchanged.

While a bird is mildly intoxicated it is attacked by a normal bird and makes no defense. This lack of response to painful stimuli is not unlike the behavior of the catatonic patient when pricked with a pin.

The abruptness with which catatonic behavior is interrupted by external stimuli and with which catatonic behavior is resumed on the cessation of external stimuli is likewise suggestive of the blocking of speech and other activities of catatonic patients.

Awkward positions are not assumed spontaneously but within certain limits of intoxication they may be imposed provided that the bird is handled gently and has not been excited by more potent stimuli.

Passive negativism or automatic resistance to being pushed is often fairly well developed but it is sometimes difficult to dissociate this from the ever present tendency to maintain equilibrium. Occasionally there is a suggestion of active negativism or a movement in the direction opposite to that of the pressure exerted by the experimenter.

Muscular tension increases in proportion to the degree of intoxication. Passive movements are therefore opposed by an increased resistance and spontaneous movements tend to become slower and more mechanical.

[9] Baruk et de Jong. L'épreuve de la bulbocapnine chez la poule. Loc. cit.

While the bird is intoxicated, it manifests no interest in food or water. Even when its beak is forced into water the bird makes no attempt to swallow but instead shakes its head as though trying to dislodge an unpleasant substance.

The period of intoxication varies from one-half to several hours according to the amount of bulbocapnine injected. Larger amounts are tolerated when the drug is given in divided doses and the catatonic state is thus maintained for a longer period than when the same amount is given in a single dose. As long as the moderate doses are not exceeded, the immobilization tends to be more stable as the size of the dose is increased.

Hyperkinetic Phenomena.

When doses larger than those suitable for the induction of a catatonic state are injected, the manifestations of intoxication change accordingly. Muscular tension increases, posture changes from a flexed type to one of hyperextension, and there is a predominance of hyperkinetic phenomena. In the following experiments, the dose was gradually increased with individual birds until some of them succumbed:

Siskin No. 4 (Crysomitris spinus). Weight 11 Gm.

Experiment no. 18.

11:06	A. M.	Injected 2 mgm. pure bulbocapnine.
11:07	"	Motionless. Head hyperextended.
11:09	"	Shaking head. Breathing more deeply. Head greatly hyperextended.
11:12	"	Vomiting and swallowing movements.
11:15	"	Sitting with eyes partially closed, head hyperextended. Picked at by control bird—aroused. Vomiting movements.
11:18	"	Sits with one wing extended—easily aroused.
11:20	"	Slight quivering of wings.
11:25	"	Less active than normal even when stimulated—will not do more than hop.
11:27	"	After repeated stimulation quivering of both wings.
11:32	"	Sits with eyes closed. Wings quivering.
11:35	"	Same.
1:30	P. M.	Less active than normal—sits with eyes closed but when approached by stick flies away—then immediately resumes quiet, sitting posture with eyes closed.

2:30 P. M. Sitting crouched in corner of cage—eyes closed.
2:55 " Hopping about—picking up seeds. Drinking.
3:15 " Apparently normal, flying about actively, quite alert, eating and drinking.

FINCH NO. 1. WEIGHT 16 GM.

EXPERIMENT NO. 5.

10:20 A. M. Injected 5 mgm. bulbocapnine hydrochlorate.
10:23 " Unable to stand. Tonic contractions both feet. Labored breathing.
10:25 " Clonic convulsive movements of wings. In making desperate attempts to move, whirls about and falls off table.
10:28 " Tonic and clonic convulsive movements of legs. Turns around on horizontal and vertical axes. Eyes open.
10:30 " Turns clockwise. Lying on back. Rhythmic movements with hyperextension of back.
10:31 " Breathing gradually more feeble. Died.

PIGEON NO. 1. WEIGHT 335 GM.

EXPERIMENT NO. 11.

3:40 P. M. Injected 40 mgm. commercial bulbocapnine. Replaced in cage—unsteady—swaying forward and backward—defecated.
3:43 " Settling down.
3:45 " Swallowing movements.
3:48 " Very ataxic—flying about cage—can scarcely stand. Hyperextension of back—resting on tail feathers—tonic spasm of extensor muscles—opisthotonus.
3:50 " Unable to stand—tries to fly but unable—lying on side.
3:53 " Flaps wings violently—unable to rise from floor.
3:55 " Flexions and extensions of legs as though trying desperately to walk—by violent flapping of wings manages to slide along the floor.
4:00 " Continues to slide along floor—flapping wings.
4:10 " Same.
4:30 " Same. Prefers dark corners.
4:40 " Movements a little less violent. No vomiting or diarrhœa observed. At times suggestion of vomiting movements. Tonic extensions of legs.
5:00 " About same—flapping wings spasmodically—sliding along floor—not able to rise or stand—lies mostly on right side.
5:30 " More quiet. Occasional spasmodic flapping of wings and sliding along but not nearly as violent.
5:40 " Remains in awkward positions on breast, with one wing hyperextended at side of box—then flies about with better coordination but still unable to rise from floor.

6:00 P. M. More quiet but apparently very tense and excitable—still unable to stand or rise above floor. Legs hyperextended most of time.
6:10 " Replaced in cage. Gets in corner and apparently unable to get out. On the next morning found dead.

PAROKEET NO. 2. WEIGHT 22 GM.

EXPERIMENT NO. 21.

11:24 A. M. Injected 3 mgm. bulbocapnine hydrochlorate.
11:25 " Unsteady. Vomiting movements.
11:30 " Vomiting movements continue.
11:35 " Watery defecation. Somewhat drowsy.
11:47 " Protests against being handled by biting and squawking.
11:58 " Injected 3 mgm. bulbocapnine.
11:59 " Sits with eyes closed—wings extended. Respirations more rapid.
12:02 P. M. Lying on floor of cage—short period convulsive movements.
12:05 " Wings and feet spread. Athetoid movements of head. Can be pushed *en bloc*. Toes flexed.
12:06 " Occasional convulsive movements of wings. Feeble slight ejaculatory sounds.
12:08 " Wings fully extended in tonic spasm.
12:12 " When stimulated tonic and clonic convulsive movements of wings and legs. Otherwise lies prostrate. Hyperextensive toes and legs. Following stimulation tonic—clonic convulsive movements.
12:16 " Toes fully flexed.
12:21 " Tonic—clonic convulsive movements on stimulation, lasting about one second.
12:27 " Spontaneous convulsive seizures lasting ½ second.
12:45 " Lying prostrate, toes flexed—no more tonic—clonic spasms on being stimulated. Eyes open.
1:05 " Same.
1:30 " Found dead.

There seems to be much uniformity with which different birds respond to bulbocapnine intoxication. Among the first signs of excessive intoxication is vomiting, usually followed in a few minutes by diarrhœa. Both vomiting and diarrhœa become violent and profuse as the dose is increased. When lethal doses are given, however, neither vomiting nor diarrhœa are observed and it seems probable therefore that these disturbances of the gastrointestinal tract afford a means of rapid elimination of the drug.

Disturbances of coordination and equilibrium also become greater as the dose is increased and when the intoxication is profound the bird is no longer able to fly, walk or even stand. Locomotion is greatly impeded also by the general muscular hyperexcitability. As the dose is increased, muscular activity passes from a state of hypertonic rigidity to that of tonic-clonic spasms which are elicited on the slightest stimulus and finally convulsive seizures occur spontaneously.

In some of the hyperkinetic phenomena there is a suggestion of the blind, impulsive and explosive behavior of catatonic patients but the type of reaction to larger amounts of the drug is not nearly so suggestive of the catatonic state as that elicited by smaller doses.

However suggestive of human catatonia the reactions of birds to bulbocapnine injections may be the experiments with animals having a well-developed neo-cortex are most illuminating. With suitable doses of bulbocapnine practically all of the phenomena observed in human catatonia can be reproduced. All mammals do not react alike and there are slight individual variations among members of the same species, but the reactions obtained experimentally are at least as consistent as those observed clinically in the human being.

The first experiments with bulbocapnine were upon the cat [10] but since then the reactions of the mouse, guinea pig, rabbit, dog and the monkey have been studied. Of these animals the mouse, the cat and the monkey are most suitable for studying experimental catatonic phenomena.

Only a few examples of mammalian reactions to bulbocapnine will be given here since de Jong and Baruk have already published similar studies in considerable detail. The amount of drug injected in any given animal was gradually increased with succeeding experiments but only occasionally was a lethal dose employed.

In some of the experiments with cats and monkeys, an attempt was made to observe the gastrointestinal functions by means of X-ray while animals were under the influence of bulbocapnine. I had already observed in the human catatonic that there is a definite tendency toward high position, hypertonicity but hypomotility,

[10] de Jong, H.: Nederlandsch Tijdschrift voor Geneeskunde, 1923, p. 794.

particularly of the colon[11] and I was hopeful that I might observe this index of vegetative nervous functions in the artificial catatonia. The results were inconclusive because of the tendency to vomiting and diarrhœa and the difficulty in getting the animals to take sufficient food and fluid while under the influence of bulbocapnine. The manifestations of the bulbocapnine intoxication were nevertheless very interesting and they will be included in this report.

CAT NO. 1. RATHER ACTIVE MALE. WEIGHT 2300 GM.

EXPERIMENT NO. 35.

May 2.	
10:00 A. M.	Injected 100 mgm. commercial bulbocapnine.
10:10 "	Mews frequently. Looks about somewhat furtively and apprehensively. Seems to be in some distress.
10:30 "	No spontaneous movements except wagging of tail. Resists any movement imposed and can be pushed along like inert mass. Always protects self against falling and when dropped always lands on feet. Maintains given awkward postures.
9:00 P. M.	Has had no food all day. Will not eat spontaneously. Swallows food and licks lips when fed with spoon. Continues to be apprehensive. Prefers dark room or to hide face in the corner.
9:30 "	Spoon fed barium mixture (barium, milk and chocolate).
10:00 "	X-rayed.
May 3.	
9:00 A. M.	X-rayed. Little spontaneous activity.
12:00 P. M.	Injected 100 mgm. commercial bulbocapnine.
9:00 P. M.	Will not eat spontaneously. Not tempted by meat.
9:30 "	X-rayed. Stimulated to approximately normal behavior by necessary manipulations. A few minutes after being returned to cage was observed licking and cleaning hind leg. Spontaneous movements gradually ceased and cat remained with hind leg poised in the air, back arched and head bent forward as though about to continue the cleaning process. This awkward position was maintained for 15 min. and was succeeded by another equally awkward position.

[11] Henry, G. W.: Some Roentgenologic Observations of Gastrointestinal Conditions Associated with Mental Disorders, Amer. Jour. Psychiat., 3: 681-695, April, 1924.

Gastrointestinal Motor Functions in Schizophrenia. Roentgenologic Observations, Amer. Jour. Psychiat., 7: 135.

CAT NO. 2. MODERATELY ACTIVE MALE. WEIGHT 2000 GM.

EXPERIMENT NO. 36.

May 7.

11:00 A. M. Ate two saucers of barium mixture spontaneously.

8:00 P. M. No food during day. Ate another barium meal.

8:30 " X-rayed.

9:00 " Injected 75 mgm. commercial bulbocapnine. Spontaneous activity gradually ceased. At end of one half hour remained in one position indefinitely. Maintained awkward positions imposed. Could be pushed *en bloc* although any movement was resisted. Marked drooling.

May 8.

10:00 A. M. Has had no food. Looks at milk awhile and after some hesitation laps up a little. Then shows no further interest but continues to lap milk when mouth is repeatedly forced into it. Tremor of jaws when mouth forced open with a spoon. Prefers to remain in one position and to be left alone. Slightly apprehensive. Withdraws head and body slightly when approached but takes no steps. Several attempts made during the day to get the cat to drink milk but will do no more than lap up a little when mouth is repeatedly forced into milk. Normal control cat resents this procedure and drinks spontaneously until satisfied. The catatonic cat looks at the milk as though recognizing something delectable but makes no attempt to drink. When mouth is forced into the milk the lips are licked as though the taste was enjoyed.

8:00 P. M. X-rayed.

May 9.

10:00 A. M. Has had no food. Drinks milk spontaneously but only after mouth has been forced into the milk.

MONKEY NO. 1. MALE.

EXPERIMENT NO. 38.

June 3.

10:00 A. M. Eats bread soaked in barium mixture.

12:40 P. M. Injected 50 mgm. commercial bulbocapnine.

12:45 " Less active. Inclined to be drowsy.

12:48 " Clasping another monkey. Definite tendency toward generalized flexion.

12:50 " Sits motionless with eyes closed, clasping other monkey.

12:51 " Aroused by approaching stick—withdraws—stands up and appears alert—then goes back to clasping other monkey.

12:55 " Sits with front legs extended and held forward. No spontaneous movements even though pushed by other monkey. Face expressionless—fixed, staring gaze.

12:57 P. M. Tapped on face. Does not move.
12:59 " Arms and hands maintained in practically any given position.
1:00 " Tendency to cling to stick with hand—then holds arm extended in unusual position after stick has been pulled away. Whines occasionally.
1:06 " Grasps stick and is pulled to cage door. Remains in awkward position with one arm fully extended. Screams.
1:09 " Grunts and whines occasionally. Blank facial expression and distant, staring gaze remains unchanged.
1:10 " Screams when face is pricked with a needle, withdraws head slightly but otherwise no movement. Continues to stand in awkward position.
1:14 " Clings tenaciously to stick.
1:15 " Banana held in front of face. Makes no attempt to eat it and apparently is not interested. After some of banana is forced into mouth there is a feeble attempt to masticate and swallow it.
1:20 " Resistance to flexion and extension of arm like that observed in catatonic patient. Observer goes to lunch leaving monkey clinging to bars of cage in awkward position.
1:45 " No change in position. Same fixed, staring gaze and expressionless face.

June 4.
10:00 A. M. Apparently normal again.
4:00 P. M. X-rayed.

June 5.
3:30 P. M. Injected 40 mgm. commercial bulbocapnine.
3:50 " X-rayed. Catatonic phenomena have reappeared. Slight drooling observed.

In the next three experiments the effects of repeated moderate doses were observed. A dog and two cats were injected at the same time. In the beginning they manifested the more common hostile relationship but this disappeared as the intoxication increased.

Dog No. 1. Very Active, Playful Male.

EXPERIMENT NO. 40.

June 17.
11:07 A. M. Injected 30 mgm. bulbocapnine.
11:15 " Less active—lying down.
11:18 " Injected 30 mgm. bulbocapnine.
11:25 " Asleep but can be aroused.
11:45 " Injected 50 mgm. bulbocapnine.
11:55 " Will not maintain given positions. Prefers to lie undisturbed—apparently sleeping.
12:11 P. M. Injected 50 mgm. bulbocapnine.

12:17 P. M. Lies in apparent prostrate condition.
12:25 " Slight shivering movements. Pupils moderately dilated. Eyes partly open.
12:30 " Injected 50 mgm. bulbocapnine. Gets up, walks to door and whines to get out.
12:35 " Hind legs fully abducted. Apparently unable to resume normal position. Maintains awkward positions.
12:50 " Pupils widely dilated. Lying on floor, clasped by cat.
1:30 " Positions of cat and dog unchanged.
1:45 " Walks slowly and unsteadily on being stimulated.
2:10 " Injected 50 mgm. bulbocapnine. P. 200 R. 36.
2:16 " Not startled by noise. Muscles flaccid.
2:40 " Unable to walk or stand. Occasional shivering movements.
3:10 " No change.

June 18.

11:50 A. M. Perhaps not quite so lively but otherwise normal.

CAT NO. 5. ACTIVE FEMALE. WEIGHT 1500 GM.

EXPERIMENT NO. 41.

June 17.

11:35 A. M. Injected 30 mgm. bulbocapnine.
11:40 " Vomits.
11:43 " Injected 50 mgm. bulbocapnine.
11:52 " Hisses at dog, protrudes nails but does not move. Hangs from back of chair with front paws when placed in this position.
12:12 P. M. Injected 50 mgm. bulbocapnine. Has maintained awkward position.
12:19 " Lies on top of dog without protest. Pupils widely dilated.
12:25 " Hisses when stepped upon by dog.
12:31 " Injected 50 mgm. bulbocapnine. Remains suspended by front paws from back of chair for 5 minutes.
12:50 " Licking lips. Moving head about. P. 216. R. 100.
12:58 " Lies on dog, clasping him—when placed in this position.
1:30 " No change in position. Slight shivering when stimulated. Lachrymation.
2:10 " Injected 50 mgm. bulbocapnine.
2:16 " Occasional slight rotatory tremor of head. Lachrymation and drooling. Easily startled by noises. Definite rigidity of muscles on passive motion.
2:25 " Still lying on dog. Withdraws head reflexly and repeatedly as nose is tickled by dog's ear. Marked drooling.
2:25 " Rotatory tremor of head more noticeable.
2:34 " Tonic—clonic extension and flexion of head. Lies with nose on dog's face, apparently unable to change position. Still suspends self from back of chair with front paws when tested.

2:50 P. M. Oscillates forward and backward when placed in erect position. Gradually sinks to floor with front legs abducted. Drooling has increased.
3:10 " No change.
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12:10 P. M. No apparent change. Will not eat or drink. Maintains stereotyped posture. Clings to cage in upright position but unable to support weight. Takes a few steps when pushed. Mews occasionally. Muscles rather rigid. No spontaneous activity. Fixed stare and expressionless face.
2:10 " No change. Fed by tube.
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9:00 A. M. Found dead.

CAT NO. 7. VERY ACTIVE MALE. WEIGHT 2000 GM.

EXPERIMENT NO. 43.

11:30 A. M. Injected 50 mgm. commercial bulbocapnine.
11:40 " Sitting down.
11:45 " Injected 25 mgm. bulbocapnine. Maintains given awkward positions. No spontaneous activities but hisses when dog approaches.
12:05 P. M. Injected 25 mgm. Fluid defecation.
12:10 " Salivation and drooling. Incontinent—fæces and urine. Pupils moderately dilated.
12:20 " Sits staring at ceiling. No spontaneous movements except occasional readjustments of ears.
12:25 " Lies with foreleg fully extended and partially abducted.
12:45 " Maintains same awkward position.
1:30 " No change in position.
3:00 " Has moved leg but otherwise no change in position. Sits drooling. Will not eat but licks lips after mouth is forced into food. Still maintains given awkward positions. Occasionally moves head slightly. Gaze fixed.
4:15 " Same position. Still drooling. Licks lips occasionally and turns head from one side to the other. Tries to move legs but unable to do more than make a start. Muscles tense. Pupils moderately dilated.
4:25 " Moves one front leg a little. Occasional generalized tremor of body.
4:30 " Gets up on legs as though about to walk but maintains awkward position without taking a step. Seems to have been blocked when about to take a step. Drooling has ceased.
5:00 " Still remains poised without having taken a step. Very slight arching of back when stroked by hand.

Reactions to Other Drugs.

Occasionally the comment has been made that similar phenomena might be elicited by the administration of other drugs and even by means of some of the more common sedative drugs. For the purpose of comparing the reactions thus obtained increasing doses of luminal, sulphonal, somnifene, harmine hydrochloricum and cannabis indica were given to a series of animals.

These experiments showed that the ordinary sedative drugs in small doses produce merely drowsiness and sleep while in larger doses they cause disturbances of equilibrium and ataxia as well as the hyperkinetic phenomena characteristic of large doses of motor excitants.

Injections of harmine in small doses also produced drowsiness and reduction of general motor activity while larger doses were followed by generalized tremors, hyperexcitability, hypertonicity, spasms and finally tonic-clonic convulsions. Occasionally an animal intoxicated by this drug shows a tendency to withdraw to a dark corner of the room.

Since the results of these experiments have already been published[12] it will not be necessary to give a detailed report of them. Of the drugs employed the closest resemblance to the experimental catatonia induced by injections of bulbocapnine was obtained by the administration of cannabis indica. For the purpose of comparing the reactions of bulbocapnine a few of the experiments with cannabis indica will now be reported.

Cannabis Indica Experiments.[13]

Hen No. 1. Weight 1500 Gm.

EXPERIMENT NO. 1.

2:20 P. M. ½ c. c. (0.2 mgm.) cannabis indica dropped into throat.
2:35 " Breathes with mouth open, occasionally caw-cawing.

[12] Henry, G. W., and de Jong, H.: A Comparative Study of the Action of Bulbocapnine and Some Other Drugs in Producing Catatonic States, Acta Psychiatrica et Neurologica, Vol. 5, p. 463.

[13] Cannabis indica was administered in solution, in capsules and in the form of pills. The action of cannabis indica or hashish on man is very well known. For the effects on animals see: Frankel, S.: Smiedeberg's Arch. 49, 1903, and Joel-Frankel: Klin. Wochenschrift, No. 37, 1926, and Pflugers, Arch. Bd. 209, Heft. 4.

2:48 P. M. Picking actively and violently at various parts of body as though bothered with lice.
2:52 " Peculiar cry as though fearful.
3:00 " Getting drowsy. Except for occasional attempts to fly to a perch has remained in one position. Easily aroused by sounds.
3:05 " Occasionally picks at body.
3:25 " Again picking at self. Flies to table occasionally and is put on floor again. Continues to caw-caw. No other spontaneous activity.
3:45 " Picking at self again.

DUCK NO. 1. WEIGHT 1300 GM.

EXPERIMENT NO. 6.

12:15 P. M. Given 2 c. c. (10 mgm.) cannabis indica by mouth.
12:30 " Rather quiet but excitable.
12:48 " Given an additional 2 c. c. cannabis indica by mouth.
12:50 " Picking at self.
12:55 " Twitchings of wings. Watery defecation.
1:50 " Possibly slightly less active. Given 4 c. c. (20 mgm.) by mouth.
2:15 " Possibly a little drowsy but easily aroused. Walks and flies. Drinks very little. No apparent interest in food.
3:15 " Apparently slightly drowsy but easily aroused. No interest in food or water.
3:45 " Walking about. Eating corn. Drinking water.

CAT NO. 3. WEIGHT 2500 GM. RATHER SLUGGISH AND QUITE EMACIATED.

EXPERIMENT NO. 8.

10:55 A. M. Given 40 mgm. cannabis indica by mouth in capsule.
11:15 " Given another 40 mgm. cannabis indica by mouth in capsule.
12:00 M. Possibly not quite as active.
1:00 P. M. Slow and unsteady in movements. Slight swaying when standing or walking.
2:00 " Muscles of legs rather rigid. Slight tendency to maintain unusual positions.
2:30 " Drinks milk and eats a little meat.
3:00 " Inclined to remain in one position even though awkward. Seems to start occasionally as though aroused from a dream. All spontaneous activity has practically ceased. Moves slowly and mechanically after being stimulated. Somewhat drowsy. Apparently not disturbed by loud barking of dog nearby.
3:20 " Remains in half sitting position. Gives start occasionally.
3:30 " Prefers to keep near wall. (Sits on a ledge.)

4:00 P. M. Still maintains unusual positions. For a short time stood with front paw resting on hind paw. Apparently quite unsteady—starts as though to prevent falling. Makes attempt to drink milk and to eat some meat without succeeding. Responds to caressing by raising back like normal cat. Does not purr. Remains for short time with hind legs elevated on box 6 inches high. More likely to maintain unusual positions when stimulus is minimal. Sits with paw resting on back of another cat.

4:15 " Sits dozing, occasionally giving a start and rousing.

5:00 " Same except a little more inclined to assume normal positions.

CAT NO. 9. WEIGHT 2000 GM. MODERATELY ACTIVE.

EXPERIMENT NO. 18.

11:00 A. M. Given 130 mgm. cannabis indica by mouth in capsule.

12:00 M. No spontaneous activity but reacts to dog—hair and tail erected—hissing. Will not maintain given awkward positions.

12:30 P. M. Very drowsy. Sits with nose almost touching floor.

4:30 " Drooling. Moves slowly and spontaneously.

5:00 " Drooling has ceased. Otherwise the same.

CAT NO. 9.

EXPERIMENT NO. 19.

10:55 A. M. Given 80 mgm. cannabis indica by mouth in capsule.

4:30 P. M. Definite swaying movements even when sitting down. Very little spontaneous activity. Pays little attention to presence of mice for few minutes but then tries to get at them.

DOG NO. 1. FEMALE. VERY LIVELY AND PLAYFUL. WEIGHT 3800 GM.

EXPERIMENT NO. 20.

11:35 A. M. Given 120 mgm. cannabis indica by mouth in capsule.

1:00 P. M. Less active. Sits in corner.

3:30 " Quite drowsy, lethargic and slow in movements. Will maintain awkward positions for short time but apparently because lacking in energy to move. Seems to prefer corner of room. Action tremor of extremities. When lying down eyes close wearily. Occasionally rouses with a start. Uncertain and unsteady in movements.

It appears then that certain doses of cannabis indica cause the animal to become drowsy, less active, rather indifferent to natural enemies, and to lose desire for food and drink. A sluggish, undernourished cat maintained awkward postures for a short time and an-

other cat while profoundly intoxicated manifested drooling. When sleep occurred it was fitful and the animal tended to waken with a start. The more profoundly intoxicated cats and one dog showed disturbances of equilibrium, swaying movements on standing or walking, ataxia and action tremors of extremities.

When the reactions to cannabis indica are compared with the loss of motor initiative, drooling and catalepsy induced by injections of bulbocapnine it is evident that catatonic-like reactions are only occasionally and imperfectly obtained with cannabis indica. Negativism and the striking hyperkinetic phenomena of bulbocapnine intoxication were not elicited by cannabis indica. In addition the responses to a given dose of bulbocapnine per unit weight of animal are almost constant.

Although the interesting hallucinatory experiences resulting from the ingestion of mescalin have been thoroughly investigated and fully described [14] de Jong has discovered only recently that catatonic phenomena can be produced in animals by means of this drug.[15] He experimented with certain mammals, frogs and fishes and obtained results similar to those of bulbocapnine intoxication. After the injection of from 3 to 6 mgm. of mescalinum sulfuricum into mice he observed gradual reduction of motor activity without paralysis, a posture of generalized flexion, passive and active negativism and a somewhat less active catalepsy than that of bulbocapnine intoxication. With still larger doses hyperkinetic phenomena begin to appear. The mice begin to run and jump on being touched. They finally fall down and continue to make running movements with their legs. Maximum doses produce convulsions, biting of the tongue and incontinence. Similar phenomena were observed in cats when the dose was increased to about 75 mgm. but in addition they were inclined to hide in corners or under the table, they maintained a distant, fixed gaze, they appeared anxious and they struck at apparently hallucinated objects. It was necessary to give 270 mgm. of mescalin to a monkey weighing 2½ kgm. before similar but more fully developed manifestations of

[14] Buchanan, D. N.: Meskalinrausch, Brit. J. Med. Psychol., 9: 67.

[15] de Jong, H.: Ueber Meskalin—Katatonie und die experimentelle Erzeugung von Dementia præcox—Erscheinungen, Koninklijke Adademie van Wetenschappen te Amsterdam, Proceedings, Vol. 33, No. 9, 1930.

catatonia appeared. After the injection of equivalent doses of mescalin, pigeons reacted with vomiting and negativism but frogs merely became less active, had convulsions and died.

It may be impossible to study the catatonic phenomena in human beings which might be induced by mescalin since Forster [16] has already determined that a dose as large as 800 mgm. appears to be dangerous to life. Such a dose is only a fraction of what would probably be necessary to induce an experimental catatonia.

Similar difficulties are met in trying to induce catatonic phenomena in human beings by means of bulbocapnine. In studying the action of this drug upon paralysis agitans it was found that 200 mgm. was the maximum dose which could be administered with safety.[17] Nevertheless, Henner has been bold enough to give as much as 500 mgm. and obtained marked reduction of mental and physical activity as well as cataleptic phenomena.[18]

Through some of his most recent experiments, de Jong has discovered that a state of catatonia may be induced in mammals by injections of large doses of adrenalin and also by injections of acetylcholinchlorid. An associate, J. Freud, reports that he obtains catatonic phenomena in mice by means of a phenol extract from human urine. It is especially significant that these substances are products of human metabolism. The research in this direction gives promise of very important disclosure.

Discussion.

While it is scarcely to be expected that such a complex disorder as the catatonia of human beings with its multitude of contributing factors could be reproduced by the administration of a relatively simple chemical substance the fact remains that by means of bulbocapnine injections practically all of the motor phenomena can be duplicated experimentally. It seems reasonable to assume also that

[16] Forster, E.: Selbstversuch mit Meskalin, Ztschr. f. d. ges. Neur. u. Psychiatr., 1930 Bd. 127, 1-14.

[17] de Jong, H., u. Schaltenbrand, G.: Die Wirkung des Bulbocapnins auf Paralysis Agitans und andere Tremorkranke, Klin. Wchschr. No. 1924, S. 2045.

[18] Henner, K.: L'influence de la bulbocapnine, sur l'appareil vestibulaire et le cervelet chez l'homme normal et chez l'homme malade, Recueil de travaux en l'honneur du 60e anniversaire du professeur Syllaba, Prague, 1928.

there may be sensory experiences resulting from this form of intoxication which closely resemble those of human catatonia. Peculiar visual illusions and hallucinations are regularly obtained with suitable doses of mescalin and the hallucinatory experiences associated with cannabis indica intoxication bear a strong resemblance to some forms of schizophrenic dissociation. These reactions seem to be due largely to the properties of the drugs rather than to the type of individual who is intoxicated.

In order to determine more carefully the extent to which the manifestations of human catatonia may be duplicated experimentally de Jong and Baruk have studied catatonic animals and patients at the same time. In the course of my own experimental work, I have had occasion to repeat most of their observations and I find little in what they have reported with which I might differ. They feel that human catatonia can be duplicated experimentally in practically all of its manifestations, especially when the mouse, the cat and the monkey are selected for demonstration.

The degree to which there is a loss of the capacity to initiate motor activity is illustrated by the fact that the moustache of a catatonic cat may be burned and still the animal makes no attempt to escape. Nevertheless if the same cat is pushed or stimulated sufficiently to move, it will take a few steps and then remain immobile again. It is quite able to support itself with its front paws when hung on the back of a chair and however it may be thrown into the air it always lands on its feet.

Even more remarkable is the tendency to assume unnatural or to maintain awkward postures which have been imposed. These manifestations are present at least as often as they are observed in the human catatonic. A cat may be observed to stop in the middle of an act and remain poised apparently unable to go on. When placed upon two chairs which are slowly separated the cat will support itself, until the hind and fore legs no longer span the intervening space and then when the fore legs drop to the floor the cat will remain poised in a vertical position with the hind legs still resting on the seat of one of the chairs.

Automatic resistance or passive negativism to any movement imposed is commonly observed in catatonic mammals and sometimes in birds. In addition to this resistance these animals often

present active negativism, *i. e.*, they move or take steps in a direction opposite to that of the movement imposed.

When the dose is increased a great variety of hyperkinetic phenomena is observed. A mouse which has been immobilized may suddenly run or jump a short distance and then become immobile again. The movements are rapid, of short duration, and appear automatic and mechanical. Monkeys have been observed to change facial expression, make gestures and to pose in such a way as to strongly suggest the mannerisms, grimacing and attitudinizing of patients. de Jong has even observed in monkeys over short intervals a tendency to echopraxia.

Laboratory studies have shown that both the human and bulbocapnine catatonic muscular tension is a kind of physiologic tetany characteristic of voluntary contraction. This similarity is supported by electromyographic records and by chronaxie tests.

The morbid changes in the function of the vegetative nervous system are often more marked than those of the human catatonic. Salivation and drooling are conspicuous manifestations. Polypnoea is almost constant in birds intoxicated with moderate doses of bulbocapnine. The influence of this drug on the gastrointestinal system tends to differentiate its action from whatever may be found to be responsible for human catatonia. Vomiting and diarrhœa are almost constant reactions in birds and are fairly frequently observed in other animals. The tendency in the human catatonic is more often in the opposite direction. I have demonstrated in my X-Ray studies [19] that 70 per cent of acute schizophrenics retain bariumized food in the colon for longer than five days while the chronic schizophrenic who has become readjusted at a deteriorated level shows no alteration in gastrointestinal motor functions.

The apparent indifference to food as well as the tendency to isolation closely resembles the reactions of the human catatonic. It seems as though the animal was unable to initiate and maintain the motor activity necessary to get food into the mouth but once it is placed in the mouth and tasted, mastication and swallowing proceed in a normal manner.

[19] Henry, G. W.: Schizophrenia, Assoc. Research Nerv. and Ment. Dis., Vol. 5, pp. 280-291.

These disturbances in the function of the vegetative nervous system led de Jong to believe in the beginning of his work that catatonia was chiefly a disorder of this nervous system but subsequent experiments have convinced him that the entire nervous system is affected and that the cerebral cortex is a necessary element for the production of this syndrome. This view is supported by a series of experiments performed by Schaltenbrand [20] in which he removed parts of the brain and then noted the effects of bulbocapnine intoxication. When only the cerebral hemispheres were removed and the corpora striata and the thalamus were left intact he was unable to obtain catalepsy or the posture of universal flexion following the injection of bulbocapnine. In some of his later experiments of this kind in which the animals survived for several months he was unable to confirm this observation. On the other hand Krause and de Jong [21] have removed the cerebral cortex on one side only and obtained unilateral catalepsy with subsequent bulbocapnine intoxication. The importance of the cerebral cortex in the catatonic syndrome is also suggested by the experiments reported in this paper. Animals devoid of a neo-cortex do not develop catatonic manifestations and the animals having a rudimentary neo-cortex develop these manifestations only to a limited degree while with the animals having a highly developed neocortex practically the entire catatonic syndrome of human beings can be duplicated by means of bulbocapnine injections.

If the concept of organo-toxicogenic factors in catatonia requires further evidence considerable support may be found in the numerous clinical observations of various neuropathological conditions, especially those involving the extrapyramidal system. A series of contributions on this subject have been made by Kleist [22] and an attempt has been made by Buscaino [23] to ascribe the manifestations

[20] Schaltenbrand, G.: Die Wirkung des Bulbocapnins auf Rückenmarks- und decerebriertekatzen, Pfluger's Archiv. f. d. ges. Physiologie, Bd. 209, H. 5-6, Oct., 1925.

[21] Krause, F., u. de Jong, H.: Ueber die Lokalisation einiger Motorischen Erscheinungen bei der Bulbocapnine—Katatonie, Ztschr. f. Neur. März., 1931.

[22] Kleist, K.: Die psychomotorischen Störungen und ihr Verhältnis zu den Motilitätsstörungen bei Erkrankungen der Stammganglien, Monatschr. Psychiat. u. Neurol., 1922, 52, S. 253.

[23] Buscaino, V. M.: Neue Tatsachen über die pathologische Histologie und die Pathogenese der Dementia præcox, der Amentia und der extrapyramidalen Bewegungs—störungen, Schweiz. Arch. f. Neurol. u. Psychiatr., 1924, 14, 210.

of catatonia to lesions of this system. In addition there have been a number of interesting observations of catatonic psychosis associated with some special pathological condition such as the case reported by Kant [24] which presented at different times both the hyperkinetic and the akinetic manifestations of catatonia following carbon monoxide poisoning.

Much more has been learned which relates to the pathology and pathogenesis of catatonia from the studies of the post-encephalitic parkinsonian syndromes. Although these illnesses usually can be readily differentiated the clinical manifestations are sometimes so nearly alike that a diagnosis is made with difficulty or uncertainty. Baruk [25] even reports having seen a case which presented both the catatonic and parkinsonian syndromes. The differentiation has been considered by Jelliffe [26] and by the authors to whom he refers as well as by Claude, Baruk and Thevenard.[27]

There has also been some suggestion of a relationship between sleep, narcolepsy and catatonia. Sleep is one of the more common early manifestations of bulbocapnine intoxication and if minimum doses are given this may be the more prominent manifestation. A case of narcolepsy which also developed a catatonic stupor presented by Miller [28] suggests a relationship between these conditions. He is inclined to find the seat of these disorders in the mid-brain and thalamus.

A similarly plausible aspect of catatonia is presented by Kempf [29] who regards the catatonic state as a "vicious circle of affective adaptation, followed by and retroactive with metabolic change." He calls attention to the fact that the catatonic manner of respiration is usually characterized by shallow, abdominal breathing similar to that observed in hibernating animals and in

[24] Kant, F.: Katatonie Motilitätspsychose nach Co-Vergiftung, Arch. f. Psychiatr., 1926, 78: 365.

[25] La Catatonie expérimentale par la bulbocapnine, Paris, 1930, p. 114n.

[26] The Mental Pictures in Schizophrenia and in Epidemic Encephalitis, Schizophernia, Assoc. Research Nerv. & Ment. Dis., Vol. 5, p. 205.

[27] Contribution à l'étude de la physiologie pathologique de la démence précoce catatonique, Congrès des aliénistes et neurologistes, Blois, 1927.

[28] Miller, E.: Mental Dissociation: Its Relation to Catatonia and the Mechanism of Narcolepsy, Brain, 50: 624.

[29] Kempf, E. J.: Affective-Respiratory Factors in Catatonia, Med. Jour. and Record, 131: 181-185, Feb. 19, 1930.

the auto-hypnotic hibernating adaptation of primitive people when confronted with starvation. It is not at all improbable that the human catatonic makes use of these hibernating capacities of the respiratory—circulatory segments as a form of escape from the distressing and dominating realities of life.

In view of all of these contributions to the understanding of catatonia it is difficult to dismiss the conception that this disorder is dependent in part at least upon toxic factors and upon toxic substances arising from disordered metabolism. It appears also that there is a diffuse intoxication of the nervous system with its greatest effect registered in the subcortical centers and that the cerebral cortex is necessary for the complete elaboration of catatonic phenomena.

The temporary interruption of catatonic stupor by means of cocain as observed by Berger [20] and Jacobi [21] or by means of inhalations of carbon dioxide or intravenous injections of sodium amytal [22] is consistent with the psychogenic conceptions of catatonia but since it has been demonstrated that the bulbocapnine catatonia of animals also can be interrupted by carbon dioxide inhalations the evidence is in favor of toxicogenic conceptions. Whatever may eventually be found to be true the fact that the use of these agents reestablishes contact between the patient and the external world commands further search for substances which may prolong this normal interval or perhaps bring about a permanent readjustment.

This report would be incomplete unless some consideration was given to possible psychogenic factors which might have contributed to the experimental catatonic reactions in animals. Even after practical demonstrations have been given it has been suggested that the animals might in some way have been hypnotized.

As far back as the middle of the seventeenth century, Kirscher is reported as having " obtained a cataleptic state with rigidity in

[20] Berger, H.: Zur Pathogenese des katatonischen Stupors, Münch., Med. Wochenschr., 1921, S. 448.

[21] Jacobi, A.: Die psychische Wirkung des Cocains in ihrer Bedeutung für die Psychopathologie, Arch. f. Psychiatr., 79: 383.

[22] Lorenz, W. F.: Some Observations on Catatonia, Psychiat. Quart., 4: 95. Solomon, H. C., et al.: Some Effects of the Inhalation of Carbon Dioxide and Oxygen, and of Intravenous Sodium Amytal on Certain Neuropsychiatric Conditions. Amer. Jour. Psychiat., 10: 761.

hens by forcibly holding the wings and head and making them fix upon a chalk line." The interesting experiments performed by Coriat [33] on crayfish, frogs and guinea pigs were similar to this. He was able to produce a state of catalepsy in which the animal remained motionless and rigid for a few minutes after it had been suddenly thrown on its back and sometimes held in unnatural positions. The phenomena resulting from this procedure are obviously the result of fear.

Other investigators have observed that when a beetle is turned on its back it remains motionless and apparently cataleptic with its legs sticking rigidly in the air but as soon as the observer goes away the beetle scrambles to its feet and resumes its journey. The same is true of other animals that feign death in the presence of danger. They owe their survival to this instinctive reaction to birds and beasts of prey since a dead animal is seldom attacked.[34]

In my experiments nothing was done to the animals except to inject bulbocapnine into them subcutaneously and then note the reactions to this and to the external stimuli as has been recorded. The catatonic phenomena always appeared gradually and with a consistent relationship to the dose and to the time of the injection of bulbocapnine and with no relationship to my presence or to that of any other possible hostile being. The immobility and rigidity obtained by Coriat appeared immediately and continued while he was near the animal. There were no immediate effects from the injection of bulbocapnine. He found also that the reaction was most constant and readily obtained in the crayfish or in other words the lower the animal was in the scale of evolution the more easily a motionless state could be produced. The opposite is true of bulbocapnine catatonia which is most fully developed in mammals.

The significance of experimental work of this kind appears to offer much opportunity for difference of opinion. Those who look for an organic basis for mental illness accept the results of these experiments as further evidence for their viewpoint while those who confine their interest to psychogenic factors are apt to dismiss the work as unrelated to the wish fulfillments and regressive

[33] Coriat, I. H.: The Nature of Sleep (Experiments on Animals), Jour. Abn. Psychol., 6: 339.

[34] Bramwell, J. M.: Hypnotism. Its History, Theory and Practice. Philadelphia, 1930, pp. 156, 157.

phenomena observed in human beings. The fact that birds and higher animals always react to moderate doses of bulbocapnine with a tendency to postures of universal flexion and that monkeys intoxicated by bulbocapnine have been observed in crucifixion postures, may not alter materially the psychogenic conceptions of regressions to intrauterine life or of the death and rebirth fantasies of schizophrenics.

In view of the vast amount of laboratory, experimental and clinical evidence indicating changes at both the physiological and the psychological levels of integration, it would seem more logical to regard all psychoses as having both toxicogenic and psychogenic aspects. These experiments do not show that catatonia is a toxic psychosis but the reproduction experimentally of this clinical syndrome solely by means of a known toxic agent suggests a toxic factor in this illness. After several years of clinical and experimental work I have come to the conclusion that although the factors in a mental illness prior to its onset are largely psychogenic, the presence of a psychosis means that physiological changes have already taken place, changes which tend to perpetuate the psychological manifestations. With few exceptions we have failed to interrupt this vicious circle. This is especially true of catatonia and it appears that we must now find some way of dealing with toxic factors or else the patient must remain imprisoned by his own musculature just as he did in the time of Hippocrates.

Such a conception is entirely in harmony with the teachings of Adolf Meyer [35] regarding psychobiology and psychopathology. It may be assumed, however, that at least the more progressive psychiatrists have disposed of the fiction of body versus mind and that we are ready to study more carefully the component integrations and the extent to which their disorders may contribute to a distortion of the total psychobiological integration. This venture into a much neglected field was intended as a step in this direction and was made without any expectation that toxic factors would ever be found to be wholly responsible for such a complex psychopathological condition as human catatonia.

[35] Inter-relations of the Domain of Neuropsychiatry, Arch. Neurol. & Psychiat., 8: 111.

Flournoy, H.: L'enseignement psychiatrique d'Adolf Meyer, Arch. de psychol., 20: 81.

Conclusions.

Practically all of the motor phenomena characteristic of human catatonia can be duplicated in the higher animals by means of injections of bulbocapnine in doses varying from five to ten percent of the body weight of the animal. These reactions are dependent upon the degree of development of the nervous system. The most complete catatonic pictures are obtained in mammals, particularly the mouse, the cat and the monkey. Catatonic manifestations are only imperfectly developed in birds and in the presence of external stimuli they tend to disappear. No distinctly catatonic reactions were obtained in the turtle, fish, frog, lizard or salamander. In other words the extent to which catatonic manifestations may be produced is directly related to the degree of development of the neo-cortex.

Catatonic phenomena appear gradually after the injection of bulbocapnine and the maximum effect is obtained in about one-half hour. Smaller and divided doses tend to prolong the state but the animals usually fully recover from one injection in the course of about twenty-four hours. Somewhat similar reactions have been obtained from the administration of relatively large doses of cannabis indica and mescalin.

Minimum doses of bulbocapnine produce drowsiness, reduction of motor activity, negativism and catalepsy. With moderate doses, especially in mammals, the catatonic manifestations are most fully developed. Still larger doses are followed by motor phenomena characteristic of a state of hyperexcitability and hyperkinesis which may be terminated with tonic spasms, convulsions and death. The hyperkinetic reaction to large doses of bulbocapnine is not unlike that induced by excessive doses of numerous other motor excitants.

These experiments are presented as additional evidence of toxic factors in human catatonia. The reproduction of catatonic phenomena experimentally suggests that toxic as well as psychogenic factors must be investigated before the secrets of this disease can be known.

DISCUSSION.

DR. ARMANDO FERRARO (New York, N. Y.).—It is a pleasure for me to discuss Dr. Henry's paper because of the fact that his contribution is the result of an experimental work. I personally am very much in favor of experimental work in our attempts to solve some of the most fundamental questions concerning mental pathology and therefore I always feel very happy to meet with investigations offering an experimental approach.

It is high time that psychiatry be considered on the same level as any other specialized branch of medicine and therefore it is high time that in our investigations of psychiatric problems we use the same experimental methods so frequently used in medicine.

Dr. Henry's very interesting paper deals with one organic approach to mental diseases. It deals with the toxic approach and he has presented to us some of the characteristic clinical manifestations that a toxic agent is apt to produce in animals. Dr. Henry compares and tends to identify these clinical manifestations with the same manifestations of catatonia in man. I feel, however, that he does not go as far as Baruk and de Jong, who decidedly identify experimental catatonia with catatonia in man not only in regard to the cataleptic manifestations, but also in regard to the psychic components the involvement of which according to them precedes the motor involvement.

I agree in fact with Dr. Henry as to the similarities of human and experimental catatonia and there is no doubt that we have witnessed in animals treated with bulbocapnine, the occurrence of motor phenomena identical to the motor phenomena occurring in human catatonia. I feel, however, that the identification has to be limited for the time being to the motor character of the reactions leaving out of the question the psychic components which in the animals are still unknown to us. By so limiting the problem we all see the possibility that the motor manifestations of catatonia might be reproduced by the use of an entogenous toxic agent.

The similarities of the motor phenomena in human catatonia and experimental catatonia are still enhanced by the comparison of the results following the administration of CO^2 and oxygen in human catatonia with the same results occurring in experimental pathology. We have been able in fact to submit monkeys and cats, under the influence of bulbocapnine, to the combined action of CO^2 and oxygen and witnessed the beneficial influence of the gas mixture over the motor catatonic components. The animals came out of the gas chamber, being able to temporarily walk and jump around, a quite contrasting picture with their previous cataleptic attitudes.

The similarities of catalepsy experimentally produced by the use of bulbocapnine and human catalepsy is also enhanced by the fact that Buscaino has injected as much as 100 milligrams of bulbocapnine intravenously to already catatonic patients and has produced in these patients an accentuation of their cataleptic manifestations.

The above mentioned similarities of the motor components cannot be extended, however, according to my own estimation, to the psychic components which naturally imply the action of a cortical mechanism. On the importance

of such a cortical factor Baruk and de Jong are very positive and for them, the catalepsy, the so-called negativism and the interrelation between motor and psychic elements are the expression of this cortical factor.

To explain my attitude I must recall the fact that at the New York State Psychiatric Institute my assistant, Dr. Barrera and myself, have been interested in the same experimental problem of reproducing catatonic features in animals. The purpose of our investigation has been essentially to establish if any cortical element was involved in the experimental syndrome and the extent of such a cortical factor.

For this purpose we have operated a large series of animals (cats and monkeys) to which we have removed the most various areas of the brain cortex either unilaterally or bilaterally. The results of our experiments are quite in contradiction with Schaltenbrand's, de Jong's and Baruk's conclusions to which Dr. Henry subscribes, that catalepsy is a cortical manifestation. We have in fact been able to reproduce the same motor manifestations including catalepsy, loss of motor initiative and maintenance of passive positions not only in animals deprived of one small portion of the cortex (motor area, frontal area, occipital area) but also in animals deprived of the whole cortex on one or both sides. It follows naturally that the cortex is not essential to elicit this so-called experimental catatonia.

We have gone farther in our experiments and have removed in some animals portions or all of the striatum. Up to the present moment the cases controlled histologically allow me to state that even following the removal of the cortex in addition to the removal of the neo-striatum (caudate and putamen) we have been able to reproduce catatonic manifestations in the operated animals. We also have reproduced the same clinical picture following the medial-line splitting of the mesencephalon with the subsequent interruption of the crossed neuro-spinal tract, the major efferent tract of the paleo- and neo-striatum.

We are therefore justified in stating that experimental motor catatonia induced in animals by bulbocapnine is the result of the toxic action of the drug over areas of the central nervous system which are not, however, either the cerebral cortex or the striatum. It is possible that the areas may be represented by some of the diencephalic or mesencephalic centers or even some peripheral neuro-muscular apparatus which seems to regulate the tonus and plasticity of the muscular tissue.

The fact remains, however, that the motor components of catatonia can be reproduced experimentally by the use of a toxic substance, a fact which clearly indicates the action of a toxic element in the same clinical picture.

I am in full agreement with Dr. Henry when he advocates the importance of the toxic factor in the determination of psychosis and I agree with him when he says that we must now find some way of dealing with toxic factors or else the patient must remain imprisoned by his own musculature.

In harmony with my own feelings I will only mention the fact that in my department at the Psychiatric Institute, in collaboration with Dr. Kilman and Dr. Strutton, experiments have been carried on for the last six months dealing with the toxic influence of constipation and intestinal obstruction over the

brain structures and the influence of various toxic substances which are present in the gastrointestinal tract over the behavior of animals and over the brain structures with the intent of reproducing changes comparable to the ones that we find in cases of non-complicated dementia præcox. This is not the place to discuss the details of our plan of work, but I am glad of the opportunity to state that our preliminary investigation has been quite successful and that we have already detected histological changes of definite significance.

The fact that in the particular case of experimental catatonia the cerebral cortex is not essential in the determination of the motor components does not detract at all from the importance of Dr. Henry's contribution who courageously states his own opinion concerning the importance of organic factors in the development of psychoses. On the contrary, Dr. Henry's contribution, besides its intrinsic value, represents a warning for the necessity of putting psychiatry on the same scientific experimental level as any other branch of medicine, and such an attempt which he has already started deserves all our appreciation.

DR. LANGENSTRASS.—We have produced experimental catatonia in cats. Bulbocapnine was given by subcutaneous injection and worked very promptly in each instance. In our experience the catatonia-like condition developed ten to twenty minutes after injection of the drug and persisted then for about seven hours. Inhalation of mixtures of carbon dioxide and oxygen produced regularly a transient disappearance of the catatonic symptoms. Inhalations of pure oxygen, however, did not seem to have any noticeable effect on the bulbocapnine-syndrome. I wish to emphasize that our experience is only limited and does not permit us to draw any definite conclusions.

FUTURE PUBLIC EDUCATION IN MENTAL HYGIENE.*

By SANGER BROWN, II, M. D.,
Assistant Commissioner, New York State Department of Mental Hygiene.

To influence mankind the principles underlying any new movement must be generally known. If the movement is of bizarre nature, no thought need be given to publicity; it will be universally discussed. But movements for human betterment need support and direction. Public education in preventive medicine whereby knowledge of the most important physical diseases has become almost universal, shows what may be accomplished. The need of fresh air, pure food and pure water is recognized everywhere in civilized countries. Maternal and infant welfare, school hygiene and industrial hygiene are widely practiced. The extent to which this movement has improved the health of the nation is incalculable. The practice of medicine can scarcely be thought of without such preventive facilities as quarantine, vaccination, sanitation, food laws and other regulations which exist today.

Mental hygiene has reached the time when a similar campaign is necessary. Its applications are quite as broad as those of public health, more significant in fact in the ultimate values in life. Child training, dependency, penology, and many other problems, as well as the care and treatment of the mentally ill fall within its scope.

Dr. C. M. Hincks in an address at the First International Congress on Mental Hygiene in Washington in May, 1930, rightly stated that the problem of public education in mental hygiene consisted of emphasis on two aspects; the first comprises the securing of public understanding and backing for the various phases of country-wide mental hygiene progress; the second, and in many ways the most important, is the instruction of the public in the principles of mental hygiene for self-application. One is public, the other personal. While a number of people are well informed on this subject, education in mental hygiene for the great mass of the people is practically virgin soil.

*Read at the eighty-seventh annual meeting of The American Psychiatric Association, Toronto, Canada, June 2, 1931.

What Should be Taught.

What should be taught in mental hygiene? There are certain essentials which the public should know. The commoner forms of mental maladjustments and conflicts which so many persons experience, should be made clear. Such conditions as anxiety states, fears, depression, emotional instability, mental exhaustion, irritability, sensitiveness, undue aversions and other mental difficulties are subjects for public education.

While it is not feasible to discuss these conditions in complete detail, much valuable information may be given. The public may be told that these symptoms do not generally arise from physical disease alone; that they are not solely inherited; that they indicate a lack of harmony in mental life; and that they are susceptible of treatment. The public may be told that through self-education in some instances and treatment by a skilled physician in others, much benefit may result.

These topics should be dealt with in a clear and simple way. A symptom such as mental depression may be dealt with in a radio talk, lecture or pamphlet so that certain causes may be stated, certain symptoms indicated and underlying principles of treatment suggested. Everything known cannot be told about depression in a radio talk, a fact which is equally true of diphtheria, but information can be given which will lead to rational treatment and the abandonment of unwise methods.

In child guidance the need of parental education is well recognized. The causes and management of such universal conditions in children as temper tantrums, nervousness, backwardness, faulty habits and various other symptoms should be brought before the public. Pamphlets on child guidance distributed by the United States Children's Bureau and by the National Committee for Mental Hygiene and other organizations, represent an important move in public education. Several volumes printed by the Commonwealth Fund in the interests of child guidance are widely read. In fact, there are dozens of books on certain phases of child guidance and management.

Many other special fields in mental hygiene should be better known. Psychiatry in industry, which is comparable to public health work in industry, should be widely understood. The mean-

ing of mental hygiene in secondary and college education should be known. The application of psychiatry to penology requires a campaign to bring about its general adoption. The implications of mental hygiene in relief work for dependent persons should be put to practical use.

Who Should be Taught Mental Hygiene?

Who should be taught mental hygiene? Its teaching should pervade society and its principles should be known to the man on the street. While the important facts of life filter gradually through from the more enlightened to the less informed and may eventually reach the ignorant, this process may be hastened by an educational campaign.

Special groups need special education. The entire medical profession should be more familiar with mental hygiene principles. In the future many psychoneuroses now masquerading as physical diseases will be recognized as symptoms of mental maladjustment. The importance of mental factors in cardiac, gastro-intestinal, respiratory and other diseases will be better understood. The medical profession, however, cannot alone take responsibility for preventive work in this field. Well qualified specialists should direct it, but non-medical persons will have to carry a share of the work of public education.

Methods should be worked out by which the simpler principles of mental hygiene are taught in the elementary schools. This measure, possibly more than any other, would firmly introduce the philosophy of mental hygiene into the everyday life of the entire population. In a mental hygiene survey of Boston, Dr. George H. Preston reports that a beginning has been made in teaching mental hygiene in some of the public schools in that city. Similar movements are mentioned elsewhere. A textbook embodying these principles for use in schools should be prepared.

How Should Knowledge of Mental Hygiene be Disseminated?

How should knowledge of mental hygiene be disseminated? Like other topics of public interest, it is disseminated spontaneously to some extent without any publicity. Novels appear from time

to time with some problems of child guidance as the theme. Articles on mental hygiene, sometimes by eminent specialists, appear in newspapers. Spontaneous methods of public education, however, serve a limited purpose. Press articles have more news than educational value. The supporters of public health would not wish to leave their campaign to news items. A carefully planned educational campaign by authoritative persons has continuity, gives facts which become accepted knowledge and has the confidence of the public.

Public Education in the New York State Department of Mental Hygiene.

A number of methods of disseminating mental hygiene information have been used by the New York State Department of Mental Hygiene with a view to informing the public of important facts regarding the nature, symptoms and treatment of mental defect and mental disabilities and handicaps. In this program, up to the present, the means used have been radio talks, the distribution of leaflets and pamphlets, articles in newspapers, a mental hygiene bulletin, and public addresses.

In March, 1931, a new radio station was started in Albany, Station WOKO, and a period of five minutes one evening a week has since been assigned free to the Department of Mental Hygiene. The mental hygiene talks are of about 600 words. They are written by various members of the department, by the superintendents of the institutions and by physicians and others on the staff. Each radio talk is mimeographed, and copies are sent to about 600 newspapers in the state on the day it is given over the radio. It is likewise sent to a few special papers like the "United States Daily."

The talks deal with one particular subject only. Some of these topics are: Play for Mental Health; The Causes of Mental Disease; The Quest for Mental Health; Pull Yourself Together; Emotions and Health; Clear Thinking and Mental Health; The Nervous Child; Food for the Mind; and others. Most of these talks have been published in newspapers and at times have attracted editorial comment. The talks would be well worth while if only for the literature they make available for distribution.

Another method of public education is through leaflets and pamphlets. In 1927 the Department of Mental Hygiene began to

prepare certain leaflets for general distribution. These leaflets are of pocket size and contain about 400 to 500 words each. Some of the titles are as follows: Your Emotions; Behavior Patterns; Accepting Life; Our Children; Sacrifice of the Children; Do Children Inherit Bad Conduct?; Facing Difficulties; Face the Future; The Age Misnamed Dangerous; Security and Mental Health; Mental Depression; Mental Disease Not a Disgrace; Our Social Inheritance.

Requests for these leaflets have been surprisingly numerous. In round numbers 47,000 copies of the leaflet, "Your Emotions," have been distributed, 58,000 of "Sacrifice of the Children," 50,000 of "Our Children," and so on. In all, over 518,000 have been distributed up to date.

Aside from these small leaflets, pamphlets of three or four pages of printed matter have been prepared. The department now has eight such pamphlets. The titles are as follows: Parental Guidance; Epilepsy and Its Treatment; Behavior; Habit Training; The Treatment of Enuresis; Character Training; The Prevention of Mental and Nervous Diseases; and What may be Hoped for in the Prevention of Mental Disease. The pamphlets have a less general distribution than the leaflets, 16,000 having been distributed of "Epilepsy and Its Treatment," and 23,000 of "Parental Guidance."

To contrast this distribution with public health literature, the New York State Department of Health distributes more than twice the above totals in one year. It has an assortment of over fifty different leaflets and pamphlets and longer articles. For the year 1930 the Division of Maternal and Child Welfare distributed 403,508 pieces of literature in all. In addition, 455,676 pamphlets, etc., were distributed by the Department of Health on such topics as contagious diseases, tuberculosis, cancer control, etc., making a total of over one million pieces of literature yearly.

The section dealing with social hygiene or venereal diseases of the New York State Department of Health in addition to distributing literature, employed four full-time lecturers who delivered lectures before 115,000 persons in 1930.

In October, 1930, the Department of Mental Hygiene started a bulletin, "Mental Hygiene News." This bulletin of four pages, published monthly and distributed free, consists, as a rule, of two

case histories of about one thousand words each, one on child guidance and one on a state hospital case, illustrating certain phases of treatment; an editorial of five hundred words on some topic of general interest; news of the department, general news items and personal items.

Mental Hygiene News has been sent to state and county health officers, county health nurses, school teachers, social workers and to the state institutions under the Department of Mental Hygiene. The initial circulation was 1000 and the circulation six months later was 1900. There have been numerous requests for it, some from abroad. Health News, a similar bulletin, published by the New York State Department of Health, is mailed weekly to about 20,000 persons.

Lectures on mental hygiene often draw large audiences. Their objective should be to bring about some active mental hygiene work in the locality, such as the establishment of a psychiatric ward in a general hospital, the organizing of a child guidance clinic, the employment of a psychiatric social worker, establishment of special classes in schools, or other mental hygiene activity.

Organization and Recommendations.

A number of other states either through department or committees have used the methods outlined above for public education in mental hygiene. The National Committee for Mental Hygiene has a broad program for public education.

What organization is desirable for carrying on this work? Every up-to-date department of health whether state, city or county, distributes public health literature, edits a health bulletin, sponsors lectures on cancer, tuberculosis and other diseases, conducts a campaign for pure food and water and other activities in preventive medicine.

The following recommendations are offered in respect to mental hygiene:

Heads of departments, superintendents of institutions, professors of psychiatry and directors of mental hygiene societies should establish the necessary organization in their departments for public education. The movement will then develop as it has in preventive medicine. Organized in this way it will be under the control of authorized persons.

In large mental hygiene departments there should be a separate bureau assigned to the work. Work of such a bureau should consist of preparing and distributing literature, radio talks, the organizing of lectures, etc. This work cannot be done by persons responsible for other duties. To write even a radio talk or small pamphlet requires time and care. Psychiatrists who write scientific articles cannot always be expected to produce, without experience, satisfactory articles for popular education.

Institutions for mental diseases should add a staff for community work and clinics to meet increasing demands. This staff should direct mental hygiene education not as a separate or independent activity, but as one of the activities of the institution. Anyone organizing the various phases of community work which institutions should do, may well read Dr. Adolf Meyer's address given at the First National Congress for Mental Hygiene in Washington in May, 1930, in which he insists upon an integration of all of these various phases of the work as a part of the general psychiatric activities of the institution.

Actual demonstration is in itself a highly important factor in public education. Community clinics held in general hospitals, schools, courts and elsewhere, are of broad educational value.

What results may be expected in mental hygiene education? While a contribution to the philosophy of life, mental hygiene is not a complete philosophy in itself for most persons. Different views are held on international relations, marriage, sex life, crime, education of the young, and other topics. These views depend upon tradition, religious background, the influence of accepted beliefs, parental teachings and other sources. Many of these traditional beliefs, the result of ripe racial experience, are of inestimable value to mankind. But recasting some of them is highly necessary. Changes, modifications and complete rejection of some traditions is sure to come, and the known facts of mental hygiene should influence opinions in these fields.

Psychiatry has thrown much light upon the functions of the mind. It has shown influences which go to make up character. It has indicated various factors in the development of intelligence and in what constitutes judgment. It has pointed out how environment, and particular forms of experience and training modify mental traits. It has emphasized the importance of sub-conscious

mental activity. It has shown how unhealthy tendencies such as the repression of emotions, rationalizing rather than facing facts and long-continued mental conflicts are the beginnings of mental maladjustments and at times mental diseases. This knowledge, if rightly applied, should reduce mental maladjustments which lead to psychoneuroses, mental diseases and other incapacities and handicaps.

The teachings of mental hygiene are not beyond the comprehension of most persons. It should be understood, so that when these difficulties are seen in a relative, a friend, an associate or a child, there may be a recognition of what should be done. To appreciate minor mental deviations in one's self is always more difficult, but this is also possible. The task of disseminating mental hygiene rests with the supporters of the mental hygiene movement. This paper is offered, not that it contributes many new facts to persons in this audience, but that the subject may be given the consideration it deserves.

DISCUSSION.

Dr. A. H. Desloges (Montreal, Canada).—*Mr. President, Ladies and Gentlemen:* Dr. Brown has pretty well covered the question of public education.

Stimulated by Dr. Hincks, the director of the Canadian National Committee for Mental Hygiene, we started to organize mental hygiene in the City of Montreal about two years ago. Our aim was to tackle the problem in the schools first of all. It was, therefore, necessary to obtain the co-operation of the different school boards, and of the universities.

But, before undertaking to educate the general public, we thought it wise to educate first the physicians. Perhaps my confrères will not like to hear me say that very few practitioners know the meaning of mental hygiene. In fact, I have not seen very many definitions which I would myself be ready to accept. The problem is a complex one. We wished to impart to our confrères the idea that a child could be backward not only on account of a mental defect, but also on account of a physical infection or affection.

All the children in our schools undergo a medical examination. When a child shows a physical defect, his parents are informed and given the proper advice, for example to have the child's tonsils removed, to take him to a specialist so that a defective eyesight or hearing may receive the proper attention, etc.

The children who do not progress normally are submitted to a mental examination. Sometimes the lack of progress is due to a focal infection;

such a cause being removed, the child will then begin to progress normally. In other cases the cause is mental, and the child is submitted to a Binet-Simon Stanford Test. According to the result of the examination, he is given a mental age, and directed either to special classes or, when there is a need for hospitalization, to an institution for the feebleminded. Special classes are few in our Province, the teachers not being prepared for that kind of teaching, but we expect to open several of these classes in September. For the feebleminded we have only one institution, of a capacity of five hundred. We would need three of a capacity of one thousand.

I will give you a few figures pertaining to the work done in Montreal from September, 1929, to May, 1931. Sixty-five schools were visited. The total population in these schools is 56,444; but it is to be noted that the examination was carried on in the first, second and third year in each school. The population of these classes is about 50 per cent of the total population of the 65 schools. The total school population of the City of Montreal is over 140,000—4086 children were examined; 602 were found to be normal and 3484 to be abnormal. The abnormal children were divided as follows:

2,731—retarded.
615—retarded and unstable.
138—unstable.

The psychiatrists recommended for the retarded, and the retarded and unstable, that 1089 children, who were simply backward, remain in the ordinary classes; that 304 feebleminded and imbeciles be sent to the institution for the feebleminded, and that finally two low grade feebleminded be placed at the Baie St-Paul Hospital, an institution specially adapted for that category of patients.

We are following in the steps of our friends of the United States. We still have a long way to go to accomplish as much as they have done, but we are doing our very best.

Should we limit ourselves to teach mental hygiene to the teachers and to the general public only? I will say no. I am strongly in favor of teaching mental hygiene to our legislators.

I will mention to you just a few figures regarding insanity and you will conclude with me that if the legislators had been careful about allowing companies to sell stock that is only water, there would probably be fewer insane people now. "Humbug" speculation is one of the causes of many breakdowns. During the year 1929 there were 7552 insane patients in the hospitals for the insane of the Province of Quebec. The increase that year was 416, about 5½ per cent. Last year there were 8458 patients in our institutions, an increase of 906 patients over the previous year, *i. e.*, 14 per cent. This increase is certainly due to the present general depression. I repeat that our legislators should be taught all about mental hygiene and insanity, in order to protect the public, and also give us the institutions

which are needed to segregate those who cannot have their liberty, and those who would benefit by the treatment provided.

If we have been able to accomplish something here, it has been owing to the cooperation of several of the states of the Union. I will mention Massachusetts particularly, because I have received the most wonderful help from Dr. Kline, the Commissioner of the Hospitals for the Insane of Massachusetts, and past-president of this Association. I will also mention the National Committee for Mental Hygiene of New York.

Gentleman, I congratulate Dr. Brown again for his very interesting paper.

DR. C. M. HINCKS (New York, N. Y.).—I should like to thank Dr. Sanger Brown for his excellent address. It has been stimulating, practical, and full of very useful suggestions.

After all, to insure mental hygiene progress, we must have an informed and sympathetic public, and therefore we need education. It seems to me that there are three outstanding objectives that we should seek in the public educational program. They have been mentioned by Dr. Brown. One is to dissipate ignorance and to overcome prejudice with regard to mental disorders, so that early treatment will be possible.

We know that when the public looks upon mental disease as a disgrace, when there is a tendency to hide the fact of the existence of a mental case, then there follows lamentable delay in seeking early treatment, when treatment can be of greatest benefit.

The second objective that I want to emphasize was brought out particularly by Dr. Desloges; and that is to interest the public in mental hygiene activities, so that governments and official bodies will spend public funds reasonably freely for needed enterprises.

We know that funds are spent grudgingly when the public is not interested. Therefore, we must see to it that the public becomes interested and informed in this field.

The third objective has been effectively set forth by Dr. Brown; and that is the laying of a foundation for a preventative program, by getting the public to apply to themselves mental hygiene principles for the conservation of mental health.

I want to congratulate those states with departments of mental hygiene that are embarking upon educational programs. Particularly I would like to mention the State of New York, where Dr. Sanger Brown is doing such excellent work with his confrères; the State of Massachusetts; Quebec under the very dynamic leadership of Dr. Desloges; the splendid work here in Ontario with Dr. McGhie, Dr. English, and their confrères; and indeed the work in many provinces of Canada and in many states of the Union. The mental hygiene societies can cooperate in this work.

In conclusion I would like to say that it is absolutely essential that we get the cooperation of the medical profession, of nurses, of social workers, of teachers, of the clergy, of all those who come into close contact with

their fellows, of all those who mould public opinion. And finally, I have this conviction: That the very genius of the mental hygiene movement lies in ability to win popular support, to give the public a sense of partnership with scientific leaders in promoting health work.

DR. J. L. MCCARTNEY (Hartford, Conn.).—In this subject of educating the public, I think the fact is sometimes lost sight of that the child by the time he reaches school has already had his character pretty well formed.

As an experiment, the Bureau of Mental Hygiene in Connecticut has launched a program, starting this year, to educate parents. The time to reach parents is when they are most interested, and that is usually after the child has just arrived. Consequently, we have started to send out with each birth certificate a chart correlating the various accepted "normal" developments from birth to 21 years, physical as well as emotional, intellectual and social.

We do not know what the response will be, but in Connecticut there are about 30,000 children born each year, and our plan is to send this chart of normal development to each of these new parents.

We hope thus to get the parent to expect normal development, and to think of the child's development not only from the physical but from the intellectual and emotional angle as well. And we hope that when any problem does develop, they will immediately get in touch with the proper authorities and thereby get the advice that is necessary.

DR. CLARENCE PIERSON (Pineville, La.).—Mr. President, I have greatly enjoyed the address of Dr. Sanger Brown. He has a wonderful organization in the great State of New York and is doing splendid work.

In Louisiana we have underway a program, which I thought might be of interest to you, as it is something new with us. In the first place, in order to secure better public appreciation and understanding of the work that is being done by our state institutions, we must let the people know that these institutions are open to public inspection at all times.

We should acquaint the public with the value of our neuro-psychiatric clinics and endeavor through the institutional staff conducting the clinics to bring our institutions into closer contact with the public. Our neuro-psychiatric clinics are free, and are conducted off the premises of the hospital so as to encourage a readier and more general public response.

The next aid in this work is the development and dissemination of mental hygiene principles. We invite the various educational institutions of our state, especially the normal schools and colleges that educate the teachers, to bring to our clinics and to our hospital, their classes in sociology, psychology and abnormal psychology. They usually devote Saturdays to these visits. In this way these students get valuable first hand information concerning the mental hospital and its work, and drop the prejudices which have come down from the old hospital system and from the old almshouse days. They get new ideas of modern methods of care and treatment

in the hospital, and a mutually helpful relationship arises between the college professor, the hospital itself and the student body. These young students, men and women, constitute 85 per cent of the future teaching body of our state.

While at these clinics the visiting students learn something of the classification of mental diseases and of the duties of the hospital departmental heads. All forms of diseases, mental and physical, are discussed. Laboratory reports are explained and demonstrated, and from these experiences, they better appreciate the lectures that they have been hearing while in attendance at the various colleges.

This program is having a good influence. It is bringing the hospital in closer contact with the public and enables it to function not only as a service but also as a teaching center. This spring we have had several hundred students from the various colleges visit the hospital for the purposes referred to. These young people are broadening their vision and developing new professional interests. Some become more interested in laboratory work, some in sociological work, some in psychiatry and some in neurology.

In connection with this educational undertaking we are planning through our social service workers to visit the counties, to come in contact with our paroled patients, to obtain the interest and cooperation of school teachers and arouse a greater interest in the possibilities of mental hygiene.

Notes and Comment.

Dr. W. H. Hattie.—It is with great regret that we record the death early in December of Dr. W. H. Hattie of Halifax, Nova Scotia. Dr. Hattie was one of the pioneers in mental hygiene and psychiatry in the Maritime Provinces, and for many years was Superintendent of the Provincial Hospital of Nova Scotia at Dartmouth. Dr. Hattie was a leader in all public health activities and had long been a member of the Faculty of Dalhousie University as Professor of Mental Diseases. He was a life member of The American Psychiatric Association. A memorial notice of Dr. Hattie's life and work will be published in a later issue of the Journal.

Pellagra in the United States.—This disease is of particular interest to the psychiatrist because of the fact that mental disturbance with deterioration has been regarded as one of the three cardinal symptoms, along with an exfoliative dermatitis and gastro-intestinal involvement.

Since 1916 the United States Public Health Service has conducted studies in the southern states with reference to the prevalence and characteristics, the endemic and possible epidemic nature of the malady. Some of the findings are recapitulated by Wheeler in *A Note on the History of Pellagra in the United States* (Public Health Reports, Reprint No. 1510, Sept. 18, 1931).

Although it has been assumed that cases of pellagra were infrequent in the South prior to the "explosive outbreak" of 1907-1908, it is interesting to note that Babcock (1912) concluded from a study of the case histories of the South Carolina State Hospital for the Insane, and other sources, that the disease had been continuously present in South Carolina since 1828. Wheeler thus sums up the information concerning pellagra generally available to medical students prior to 1908: "That it is a disease of unknown or uncertain etiology, occurring in Italy and a few other places in Southern Europe; that it involves the cutaneous, digestive and nervous systems, producing a classical and essential diagnostic

triad—dermatitis, diarrhœa and dementia; and last, but by no means least stressed, that it did not occur in the United States." Osler in the seventh edition of his *Principles and Practice of Medicine* states that pellagra "has not been observed in the United States." In the eighth edition appears the corrected statement that "it has probably been present in the South for 50 years."

Significant are Wheeler's views on the "diagnostic triad." This symptom group is "of rather infrequent occurrence when all types of endemic pellagra are considered. Such a combination of symptoms represents an advanced stage and is rarely encountered except in some of the more severe types." Of 421 field cases, while all showed characteristic skin lesions, 80.5 per cent presented no intestinal disturbance. Of 876 field cases with dermatitis, only 1.4 per cent showed mental symptoms "of a major order which might be considered attributable to the disease."

The results of investigations since the outbreak of 1907 lead the author to wonder "which was the more explosive in character, the actual increase in cases, or the suddenly acquired knowledge of the disease and the realization of its presence, aided by a rapidly spreading pellagraphobia and stock-taking by physicians."

NOMENCLATURE.—The second annual meeting of the National Conference on Nomenclature of Disease was held at the New York Academy of Medicine, December 14, 1931, with Dr. Haven Emerson as President. This Conference is made up of official delegates representing the American Medical Association, the American College of Surgeons, the American College of Physicians, and every other medical organization in the United States.

Dr. James V. May, Superintendent of the Boston State Hospital, as Chairman of the Committee on Statistics, represented the American Psychiatric Association, and Dr. William T. Shanahan, Superintendent of Craig Colony, Sonyea, N. Y., represented the Section on Convulsive Disorders. The classification of mental diseases and of convulsive disorders in the approved nomenclature will be that of the American Psychiatric Association, although it will be arranged somewhat differently, to conform with the classification of other diseases. The classification of mental disorders suitable to the requirements of the American Association for the Study of Mental Deficiency will also be included. It is quite

probable that the action of this Conference will lead to an international classification of diseases, which will be of inestimable value in standardizing hospital procedure and facilitating statistical studies.

It might be hoped that a thorough revision of the official classification of the American Psychiatric Association would be undertaken. There can be no doubt that such a revision is needed to make possible a satisfactory listing of mental disturbances in accordance with present views and practice. Medical officers in hospitals charged with the duty of classifying cases for statistical purposes constantly experience difficulty in forcing their cases into the fixed categories of the classification. The heading "other types" does not greatly relieve the situation. What a scientific and practically satisfactory classification should be is another question. Probably such an ideal system of mental deviations will never be achieved. Something better, however, might be made available than the present official classification, the chief value of which is that it has been in use many years, has furnished a basis of orientation and general understanding which was urgently needed and in accordance with which many thousands of cases have already been indexed for permanent record.

Association and Hospital Notes and News.

The ninth annual meeting of the American Orthopsychiatric Association will be held at the Lord Baltimore Hotel in Baltimore, Marlyand, February 18, 19, 20, 1932.

Dr. Ralph P. Truitt of Baltimore is chairman of the Committee on Arrangements.

THE ANNUAL ASSOCIATION MEETING.—The next annual meeting of the American Psychiatric Association will be held in Philadelphia at the Bellevue-Stratford Hotel from Monday, May 30, to Friday, June 3, 1932.

The Section on Convulsive Disorders will carry out its program on the first day, Monday. The regular sessions of the Association will begin as usual on Tuesday morning.

A preliminary program of the meeting will be sent to each member in due course.

OFFICERS AND COMMITTEES FOR 1931-32.—The following are the officers and committees of the Association for the current year:

William L. Russell, M. D., President.....White Plains, N. Y.
James V. May, M. D., Vice-President.....Boston, Mass.
D. S. Renner, M. D., Hon. Vice-President.....Skillman, N. J.
Clarence O. Cheney, M. D., Secretary-Treasurer.....New York, N. Y.

COUNCILLORS.

FOR THREE YEARS.

Walter M. English, M. D.....Brockville, Ont.
George M. Donohoe, M. D.....Cherokee, Ia.
William C. Sandy, M. D.....Harrisburg, Pa.
Franklin G. Ebaugh.....Denver, Colo.

FOR TWO YEARS.

Earl D. Bond, M. D.....Philadelphia, Pa.
Glenn E. Myers, M. D.....Los Angeles, Cal.
Horace G. Ripley, M. D.....Brattleboro, Vt.
Robert L. Dixon, M. D.....Lapeer, Mich.

FOR ONE YEAR.

Samuel T. Orton, M. D.....New York, N. Y.
Malcolm A. Bliss, M. D.....St. Louis, Mo.
Frank W. Robertson, M. D.....Stamford, Conn.
N. M. Owensby, M. D.....Atlanta, Ga.

AUDITORS.

FOR THREE YEARS.

Clarence M. Peirson, M. D.....Pineville, La.

FOR TWO YEARS.

Paul G. Taddiken, M. D.....Ogdensburg, N. Y.

FOR ONE YEAR.

Marcus A. Curry, M. D.....Greystone Park, N. J.

EXECUTIVE COMMITTEE.

William L. Russell, M. D.....White Plains, N. Y.
James V. May, M. D.....Boston, Mass.
Clarence O. Cheney, M. D.....New York, N. Y.
Horace G. Ripley, M. D.....Brattleboro, Vt.
William C. Sandy, M. D.....Harrisburg, Pa.

COMMITTEE IN PROGRAM.

FOR FIVE YEARS.

Samuel W. Hamilton, M. D., Chairman, 1931-32.....White Plains, N. Y.
Hugo Mella, M. D.....Lexington, Mass.

FOR ONE YEAR.

Harry C. Solomon, M. D.....Boston, Mass.
George H. Kirby, M. D.....New York, N. Y.

FOR TWO YEARS.

Lawson G. Lowrey, M. D.....New York, N. Y.
G. Kirby Collier, M. D., Vice-Chairman, 1931-32.....Rochester, N. Y.

FOR THREE YEARS.

Franklin G. Ebaugh, M. D.....Denver, Colo.
Theophile Raphael, M. D.....Detroit, Mich.

FOR FOUR YEARS.

William Malamud, M. D.....Iowa City, Ia.
Alvin T. Mathers, M. D.....Winnipeg, Man.

COMMITTEE ON ARRANGEMENTS.

Albert C. Buckley, M. D., Chairman.....Philadelphia, Pa.
James P. Sands, M. D., Vice-Chairman.....Philadelphia, Pa.
Earl D. Bond, M. D., Vice-Chairman.....Philadelphia, Pa.
LeRoy M. A. Maeder, M. D., Secretary.....Philadelphia, Pa.
Frederick H. Allen, M. D.....Philadelphia, Pa.
Everett S. Barr, M. D.....West Chester, Pa.
M. A. Burns, M. D.....Philadelphia, Pa.
Charles W. Burr, M. D.....Philadelphia, Pa.
Edward M. Green, M. D.....Harrisburg, Pa.
J. Allen Jackson, M. D.....Danville, Pa.
Henry I. Klopp, M. D.....Allentown, Pa.
Frederick H. Leavitt, M. D.....Philadelphia, Pa.
Seymour DeW. Ludlum, M. D.....Philadelphia, Pa.
Joseph B. McIver, M. D.....Philadelphia, Pa.
S. Metz Miller, M. D.....Norristown, Pa.
Clarence A. Patten, M. D.....Philadelphia, Pa.
William C. Sandy, M. D.....Harrisburg. Pa.
M. A. Tarumianz, M. D.....Farnhurst, Dela.
T. H. Weisenburg, M. D.....Philadelphia, Pa.
Nathan W. Winkleman, M. D.....Philadelphia, Pa.

COMMITTEE ON PUBLICITY.

For Five Years.

George K. Pratt, M. D., Chairman, 1931-32.....New York, N. Y.

For One Year.

A. S. Hamilton, M. D.....Minneapolis, Minn.

For Two Years.

Roger C. Swint, M. D.....Milledgeville, Ga.

For Three Years.

Clarence A. Bonner, M. D.....Hawthorne, Mass.

For Four Years.

George H. Stevenson, M. D.....Whitby, Ont.

COMMITTEE ON RESEARCH.

For Three Years.

John C. Whitehorn, M. D., Chairman, 1931-32.....Waverly, Mass.

For Two Years.

Hugo Mella, M. D.....Lexington, Mass.

FOR ONE YEAR.

Albert M. Barrett, M. D.....Ann Arbor, Mich.

FOR FIVE YEARS.

Clifford B. Farr, M. D.....East Gardner, Mass.

FOR FOUR YEARS.

Gregory Zilboorg, M. D.....New York, N. Y.

COMMITTEE ON STATISTICS.

FOR FIVE YEARS.

James V. May, M. D., Chairman, 1931-32.....Boston, Mass.
Albert M. Barrett, M. D., Vice-Chairman, 1931-32.....Ann Arbor, Mich.

FOR FOUR YEARS.

George H. Kirby, M. D.....New York, N. Y.
Frankwood E. Williams, M. D.....New York, N. Y.

FOR THREE YEARS.

C. Macfie Campbell, M. D.....Boston, Mass.
Walter L. Treadway, M. D.....Washington, D. C.

FOR TWO YEARS.

E. Stanley Abbot, M. D.....Boston, Mass.
Sanger Brown, II, M. D.....Albany, N. Y.

FOR ONE YEAR.

Phyllis Greenacre, M. D.....White Plains, N. Y.
William T. Shanahan, M. D.....Sonyea, N. Y.

COMMITTEE ON NURSING.

FOR THREE YEARS.

Daniel H. Fuller, M. D., Chairman, 1931-32.....Philadelphia, Pa.
Ross McC. Chapman, M. D.....Towson, Md.

FOR TWO YEARS.

Mortimer W. Raynor, M. D.....White Plains, N. Y.
Morgan B. Hodskins, M. D.....Palmer, Mass.

FOR ONE YEAR.

Albert Anderson, M. D.....Raleigh, N. C.
Roger C. Swint, M. D.....Milledgeville, Ga.

FOR FIVE YEARS.

Marcus A. Curry, M. D.....Greystone Park, N. J.
Paul G. Taddiken, M. D.....Ogdensburg, N. Y.

FOR FOUR YEARS.

Henry I. Klopp, M. D.....Allentown, Pa.
Arthur P. Noyes, M. D.....Howard, R. I.

COMMITTEE ON STANDARDS AND POLICIES.

FOR THREE YEARS.

Albert C. Buckley, M. D., Chairman, 1931-32.....Philadelphia, Pa.

FOR TWO YEARS.

Arthur P. Noyes, M. D.....Howard, R. I.

FOR ONE YEAR.

William A. Bryan, M. D.....Worcester, Mass.

FOR FIVE YEARS.

William J. Tiffany, M. D.....Brentwood, N. Y.

FOR FOUR YEARS.

Clarence B. Farrar, M. D.....Toronto, Ont.

COMMITTEE ON ETHICS.

FOR ONE YEAR.

Albert M. Barrett, M. D., Chairman, 1931-32.....Ann Arbor, Mich.

FOR FIVE YEARS.

George A. Johns, M. D.....St. Joseph, Mo.

FOR FOUR YEARS.

Edward N. Brush, M. D.....Baltimore, Md.

FOR THREE YEARS.

George S. Adams, M. D.....Yankton, S. D.

FOR TWO YEARS.

George H. Kirby, M. D.....New York, N. Y.

COMMITTEE ON LEGAL ASPECTS OF PSYCHIATRY.

For Two Years.

George M. Kline, M. D., Chairman, 1931-32.....Boston, Mass.
L. Vernon Briggs, M. D.....Boston, Mass.

For One Year.

Karl A. Menninger, M. D.....Topeka, Kans.
William A. White, M. D., Vice-Chairman, 1931-32.....Washington, D. C.

For Five Years.

Joseph W. Moore, M. D.....Albany, N. Y.
Herman M. Adler, M. D.....Berkeley, Cal.

For Four Years.

Winfred Overholser, M. D.....Boston, Mass.
Raymond F. C. Kieb, M. D.....Beacon, N. Y.

For Three Years.

Bernard Glueck, M. D.....New York, N. Y.
William Healey, M. D.....Boston, Mass.

COMMITTEE ON MEDICAL SERVICES.

For One Year.

William C. Sandy, M. D., Chairman, 1931-32.....Harrisburg, Pa.
Mortimer W. Raynor, M. D., Vice-Chairman, 1931-32..White Plains, N. Y.

For Two Years.

Edward A. Strecker, M. D.....Philadelphia, Pa.
Lloyd J. Thompson, M. D.....New Haven, Conn.

For Five Years.

Arthur G. Lane, M. D.....Greystone Park, N. J.
G. Kirby Collier, M. D.....Rochester, N. Y.

For Four Years.

Isaac J. Furman, M. D.....New York, N. Y.
Ransom A. Greene, M. D.....Waverly, Mass.

For Three Years.

Frederick W. Parsons, M. D.....Albany, N. Y.
Karl M. Bowman, M. D.....Boston, Mass.

COMMITTEE ON RELATIONS WITH SOCIAL SCIENCES.

For Five Years.

Harry Stack Sullivan, M. D., Chairman, 1931-32.....New York, N. Y.

For Three Years.

William A. White, M. D.....Washington, D. C.

For Two Years.

Arthur H. Ruggles, M. D.....Providence, R. I.

For One Year.

George M. Kline, M. D.....Boston, Mass.

For Four Years.

Bernard Glueck, M. D.....New York, N. Y.

COMMITTEE ON PSYCHIATRIC SOCIAL SERVICE.

For Five Years.

Howard W. Potter, M. D., Chairman, 1931-32.....New York, N. Y.

For Three Years.

Karl M. Bowman, M. D.....Boston, Mass.

For Two Years.

George S. Stevenson, M. D.....New York, N. Y.

For One Year.

J. Allen Jackson, M. D.....Danville, Pa.

For Four Years.

George M. Kline, M. D.....Boston, Mass.

COMMITTEE ON ACTIVITIES OF THE NEUROPSYCHIATRIC DIVISION OF THE VETERANS BUREAU.

For Five Years.

Glenn E. Myers, M. D., Chairman, 1931-32.....Los Angeles, Cal.

For Four Years.

George M. Kline, M. D.....Boston, Mass.

For Three Years.

Albert M. Barrett, M. D.....Ann Arbor, Mich.

FOR TWO YEARS.

Douglas A. Thom, M. D.....Boston, Mass.

FOR ONE YEAR.

William A. White, M. D.....Washington, D. C.

COMMITTEE ON REVISION OF THE CONSTITUTION.

FOR FIVE YEARS.

Earl D. Bond, M. D., Chairman, 1931-32.....Philadelphia, Pa.

FOR FOUR YEARS.

George M. Kline, M. D.....Boston, Mass.

FOR THREE YEARS.

Henry I. Klopp, M. D.....Allentown, Pa.

FOR TWO YEARS.

Richard H. Hutchings, M. D.....Utica, N. Y.

FOR ONE YEAR.

Lawson G. Lowrey, M. D.....New York, N. Y.

COMMITTEE ON PSYCHIATRY IN MEDICAL EDUCATION.

FOR FIVE YEARS.

Adolf Meyer, M. D., Chairman, 1931-3. .....Baltimore, Md.

FOR FOUR YEARS.

George H. Kirby, M. D.....New York, N. Y.

FOR THREE YEARS.

Edward A. Strecker, M. D.....Philadelphia, Pa.

FOR TWO YEARS.

C. Macfie Campbell, M. D.....Boston, Mass.

FOR ONE YEAR.

Franklin G. Ebaugh, M. D.....Denver, Colo.

SECTION ON CONVULSIVE DISORDERS.

D. S. Renner, M. D., Chairman.....Skillman, N. J.
M. B. Hodskins, M. D., Secretary.....Palmer, Mass.

MASSACHUSETTS PSYCHIATRIC SOCIETY.

A. Warren Stearns, M. D., President.....Billerica, Mass.
Winfred Overholser, M. D., Secretary-Treasurer.....Boston, Mass.
Morgan B. Hodskins, M. D., Vice-President.....Palmer, Mass.
Oscar J. Raeder, M. D., Councillor.....Boston, Mass.
Kenneth J. Tillotson, M. D., Councillor.....Waverly, Mass.

APPLICANTS FOR MEMBERSHIP AND FELLOWSHIP.—The following physicians have sent in their applications for membership or fellowship respectively, these to be passed upon by the Membership Committee and the Council of the Association at the next meeting.

MEMBERSHIP.

Allen, Simeon Carlyle, 311 Commonwealth Ave., Boston, Mass.
Allen, Truman J., Brandon, Vt.
Anthonisen, Niels L., McLean Hospital, Waverly, Mass.
Bateman, J. Fremont, Longview Hospital, Cincinnati, Ohio.
Bromberg, Walter, 239 E. 19th St., New York, N. Y.
Chopman, D. D., Spencer, W. Va.
Ciccarelli, Eugene C., 157 E. 72d St., New York, N. Y.
Couch, Mildred Warden, 145 E. 57th St., New York City, N. Y.
Cullins, John G., U. S. Veterans Hospital, No. Chicago, Ill.
Davidson, Henry A., 31 Lincoln Park, Newark, N. J.
deBellefieville, G. Lef., 96 Elmwood Place, Outremont, P. Q., Canada.
Dixon, Henry, 4200 E. 9th Ave., Denver, Colo.
Dunn, Frederick, Provo, Utah.
Epstein, Harry H., Parental Clinic, City of Trenton, N. J.
Ettleson, Abraham, Elgin State Hospital, Elgin, Ill.
Fowler, Murray Nathan, Athens State Hospital, Athens, Ohio.
Francis, K. Victor, Child Welfare Station, Iowa City, Iowa.
Galbraith, Hugh Malcolm, Bloomingdale Hospital, White Plains, N. Y.
Gilman, L. H., Indianapolis City Hospital, Indianapolis, Ind.
Gordon, J. Berkeley, N. J. State Hospital, Holmdel, N. J.
Helgesson, Uno Helmer, "Four Winds," Katonah, N. Y.
Howitt, John Rawson, Ontario Hospital, Woodstock, Ont., Canada.
Ives, Lionel M., McLean Hospital, Waverly, Mass.
Kettle, Ronald H., McLean Hospital, Waverly, Mass.
Langenstrass, Karl H., St. Elizabeth's Hospital, Washington, D. C.
LeDrew, Frederick, Boston State Hospital, Mattapan, Mass.
Lindemann, Erich, Psychopathic Hospital, Iowa City, Iowa.
MacNicoll, Wilfred T., McLean Hospital, Waverly, Mass.
Malcove, Lillian, 145 E. 57th St., New York, N. Y.
Miller, Wilbur R., Psychopathic Hospital, Iowa City, Iowa.
Montgomery, S. R., Ontario Hospital, Whitby, Ont., Canada.
Nielsen, Juul C., Norfolk, Nebr.
Pamphilon, W. M., Manhattan State Hospital, Ward's Island, N. Y.
Peppard, S. Harcourt, 165 Capitol Ave., Hartford, Conn.
Raymer, Charles, 4200 E. 9th Ave., Denver, Colo.
Raymond, Charles Stanley, 52 Marlboro Road, Waltham, Mass.
Robinson, Lindsay E., Greystone Park, N. J.
Rucker, S. T., Lynnhurst Sanitarium, Memphis, Tenn.
Schilder, Paul, 52 Gramercy Park, New York N. Y.
Schwarz, Frank W., Coatesville, Pa.
Steger, Edward Maples, 2952 Rosecrans Ave., San Diego, Cal.
Stranahan, G. Marion, 61 E. 86th St., New York, N. Y.
Victor, Simon Leonard, Manhattan State Hospital, Ward's Island, N. Y.
Warner, Helen T., 400 Washington St., Hartford, Conn.
Wilmott, Robert O., White Plains, N. Y.

FELLOWSHIP.

Beall, Charles R. F., Box 637, Atlanta, Ga.
Bogen, Eugene F., Hines, Ill.
Carmichael, Hugh Thompson, Albany Medical College, Albany, N. Y.
Colomb, Henry Octave, Central State Hospital, Pineville, La.
Gardner, Robert E., Mentor, Ohio.
Gardner, Walter P., 1068 Lowry Medical Arts Bldg., St. Paul, Minn.
Gilbert, Joseph LeRoy, 1437 Rhode Island Ave., N. W., Washington, D. C.
Herold, Ross E., Willard State Hospital, Willard, N. Y.
Parker, Z. Rita, 115 E. 61st St., New York, N. Y.
Rexford, Homer Isaac, Central Islip State Hospital, Central Islip, N. Y.
Rupp, Paul H., Box B, Wauwatosa, Wis.
Russell, Ernest Frederick, 149 E. Rock Rd., New Haven, Conn.
Sadler, William S., 533 Diversey Parkway, Chicago, Ill.
Silverberg, William V., Sheppard and Enoch Pratt Hospital, Baltimore, Md.
Uhler, Claude, Syracuse Psychopathic Hospital, Syracuse, N. Y.
Williams, David Bruce, Wyoming State Hospital, Evanston, Wyo.

The Membership Committee would be glad to receive comments from the membership regarding these applicants, and members wishing to send such comments should direct them to the Secretary, Dr. Clarence O. Cheney, 722 W. 168th St., New York, N. Y.

MR. BEERS' NEW YEAR'S GREETINGS.—In the unremitting activity of his mid-career Clifford Beers, the father of mental hygiene, has already somehow acquired the qualities of a tradition. His is a personality about which legends will cluster. The child he christened in 1909 in that private New Haven gathering of thirteen persons, attained its majority as the First International Congress on Mental Hygiene in Washington two years ago, when Mr. Beers was the central figure of the gathering of the representatives of fifty-three nations.

It is an altogether fitting thing that he should send to the members of the International Committee for Mental Hygiene, and to others throughout the world who are interested in its work, his New Year's message.

In an autograph-signed letter of eight pages Mr. Beers summarizes the work to date of the International Committee, the (U. S.) National Committee, and the American Foundation for Mental Hygiene, of all of which organizations he is Secretary.

Of particular interest is the announcement that sets of the Proceedings of the First International Congress will be ready for distribution in February:

> The Proceedings will consist of three volumes of about six or seven hundred pages each and will contain not only the 49 major papers delivered at the Congress but over 200 carefully edited discussions, representing a cross-section of world opinion on mental hygiene and related subjects.

Publication had been delayed by lack of sufficient funds, which were finally secured through a generous appropriation by the American Foundation for Mental Hygiene.

The Second International Congress will be held in Paris in 1935, and it is presumed that this meeting will establish the precedent of a five-year interval between International Congresses. It is appropriate that the Second Congress should be held in Paris, since France was the first country in Europe in which a League for Mental Hygiene was established. A Mental Hygiene Conference will be held in Paris during the coming summer, when European members of the International Committee, and others interested, will meet for a preliminary discussion of plans for the Congress of 1935.

In his message Mr. Beers comments on the satisfactory development of the plans of the American National Committee under its new General Director, Dr. C. M. Hincks, who has now been in office one year. Beginning with January, 1932, Dr. Hincks also takes over the editorship of *Mental Hygiene,* the official publication of the committee, assisted by an Advisory Board consisting of Dr. Howard W. Potter, Dr. Edward A. Strecker, Sheldon Glueck and Horatio M. Pollock.

Another significant development of the past year was the establishment of the Division of Psychiatric Education, which was referred to in the last issue of the *Journal.*

The question of budgets is a pertinent one in these years of financial depression, and it is gratifying to learn that the immediate future both of the National Committee and of the American Foundation are well provided for. To quote Mr. Beers:

> This account of the finances of the National Committee and of The American Foundation for Mental Hygiene does not mean, of course, that these organizations are not in need of further help and support, but it does mean that their work can be more surely financed during the coming two years than can the work of many other similar organizations in this country, which, because of the financial depression, are unfortunately finding it difficult to continue some of their basic activities.

The *Journal* feels sure that it is expressing the unanimous sentiment of its readers in reciprocating most heartily the good wishes with which Mr. Beers concludes his New Year's message to mental hygienists throughout the world.

NEW YORK'S CRIME CLINIC.—With the opening of the New Year the city of New York finds itself provided with a psychiatric clinic in its Criminal Courts building, where every prisoner convicted of a felony will be examined and a psychiatric report will be submitted to the court before sentence is pronounced.

The new clinic is a part of the Department of Hospitals, of which Dr. J. G. William Greeff is Director, and will be under the immediate charge of Dr. Menas S. Gregory, Director of the Division of Psychiatry, who has for many years conducted the Psychopathic Division of Bellevue Hospital. This development marks a notable advance in the science of criminology in New York.

Judge Collins, at the opening ceremonies, declared the inauguration of the clinic to be "the greatest step in criminal justice in the history of the State." In the opinion of Mayor Walker it is "probably the most economic approach to the curtailment of the cost of criminal justice that the State has ever undertaken."

Dr. Gregory promises to supply at a later date for publication some account of the workings of the new clinic.

DR. TIFFANY HONORED.—New York's newest mental hospital, destined to be the largest of its kind in the world, was opened in November last with Dr. William J. Tiffany as superintendent. This thirty million dollar institution, situated near Brentwood, Long Island, to serve the metropolitan area, has been given a distinguished and honorable name. It is called the Pilgrim State Hospital. In this very happy and appropriate way have been recognized the services to the State of New York and the significance to American psychiatry of Dr. Charles W. Pilgrim, formerly chairman to the State Hospital Commission, for a long period superintendent of the Hudson River State Hospital, President of the American Psychiatric Association in 1911, and now Physician-in-Charge of Dr. MacDonald's House at Central Valley.

Dr. Tiffany's training and experience eminently fit him for this important post. He first entered the New York State Hospital Service in 1904 as clinical assistant at the Binghamton State Hospital. He has also served on the staffs of the Matteawan State Hospital and the King's Park State Hospital, of which latter institution he became superintendent in 1928.

DR. ELISHA H. COHOON.—The Massachusetts Psychiatric Society has adopted and caused to be spread upon its records the following:

RESOLUTIONS ON THE DEATH OF ELISHA H. COHOON.

By the death of Dr. Elisha H. Cohoon there has been lost to psychiatric service, to numberless patients, to numerous employees and to loving friends a great administrator, a tireless servant, a justly respected employer and a most devoted friend.

Dr. Cohoon, born in Nova Scotia 57 years ago, graduated from Acadia University, N. S., and received his medical education in Baltimore, Md., graduating from the College of Physicians and Surgeons. He joined the staff of the State Hospital at Mt. Pleasant, Iowa, for three years, then served a year at the Colorado State Hospital at Pueblo.

The next seven years he spent at the State Hospital at Howard, R. I., where he firmly established a reputation as a successful executive. He was called to serve as administrator of the Boston Psychopathic Hospital for a period of two years, leaving there to accept the superintendency of the Medfield State Hospital, Medfield, on April 1, 1917.

To Medfield Dr. Cohoon brought a keen knowledge of psychiatry, an understanding interest in the welfare of his patients, and a deep sense of justice to all, both patients and employees. His courteous attitude toward the public was another distinguishing mark of a truly great man. Medfield State Hospital received the benefit of numerous carefully planned and executed innovations and improvements for the care and welfare of its patients. Dr. Cohoon introduced new facilities for treatment according to the best of methods and established many forms of physical comforts and entertainments for his patients. His employees knew him as a gracious employer, tolerant, and, above all, just in every respect. In the community much of his time was freely given to addresses before civic organizations of all sorts. Especial attention was given to out-patient clinics in nearby towns.

After a lingering illness, death claimed Dr. Cohoon on July 21, 1931, while a patient at a Boston hospital. He was a member of the Massachusetts Medical Society, The American Psychiatric Association, and of the New England Society of Psychiatry. At the time of his death Dr. Cohoon was president of the Massachusetts Psychiatric Society. The members of this society who knew him so well and loved him so genuinely have sustained a severe loss of counsellor and friend. This society may well be proud of his record, mourn his loss and, in reverence, pay its respects to the passing into the Great Adventure of a truly great physician.

PSYCHIATRY AND PRISON PROBLEMS.—A luncheon conference called by the National Committee on Prisons and Prison Labor to discuss the relationship of psychiatry to the problem of the adult prisoner was held in Toronto, Canada, on June 5, 1931, during the sessions of the American Psychiatric Association.

The National Committee on Prisons and Prison Labor was represented by Dr. E. Stagg Whitin, Chairman of the Executive Council. The guests included officials of federal and state penal institutions, Departments of Correction, Public Welfare Commissions, mental hospitals and clinics, criminologists and members of the American Psychiatric Association.

Dr. Vernon C. Branham, Deputy Commissioner of Correction of the State of New York, presided at the Conference and opened the discussion by emphasizing the need of further extension of psychiatric teaching and practice in the correctional field. "Crime has become perhaps the foremost topic of interest in this nation. It has demoralized the social structure to such an extent that serious thought and attention of the best minds throughout the country are required to combat it. Psychiatry, on account of its peculiar approach to the problems of the individual, necessarily has a very important rôle to play in the question of crime."

Dr. Whitin outlined the progress which has been made since the introduction of psychiatry in prisons through the leadership and support of Dr. Walter B. James, Dr. Thomas W. Salmon, Dr. William L. Russell and others.

Dr. Frederick J. Farnell, Chairman of the Rhode Island State Public Welfare Commission, described the procedure in that state, whereby every prisoner sentenced to the penitentiary is given complete physical, mental and sociological examinations, in a systematic study of the causes of criminal behavior. Mr. Thomas E. Murphy, Secretary of the Commission, observed that if all the offenders under the Mann Act and the Volstead Act were released, American prisons would be as empty as those of Canada. "If we want to empty our prisons we must halt our legislators."

Dr. Russell reviewed the developments in criminology during recent years and referred to the work done by special committees of the American Psychiatric Association. He suggested the desirability of closer cooperation by all the agencies interested.

Following a general discussion the Conference adopted a resolution that a Committee on Cooperation, with Dr. Whitin as Chairman, be appointed with the advice of Dr. Russell, the incoming President of the American Psychiatric Association.

AMERICAN OCCUPATIONAL THERAPY ASSOCIATION.—The fifteenth meeting of the American Occupational Therapy Association held in Toronto, Ontario, September 28-30, 1931, was a notable one. The total registration was 268, of whom 178 were members of the Association. This was the first meeting of the Association held outside of the United States, and the Canadians contributed much to its success, both by their participation in the program and by their charming hospitality.

The program included some 20 papers covering all aspects of the occupational therapy field, grouped under the following headings:

General hospitals.
Curative workshops and field service.
Mental hospitals.
Out-patient and social services.
Orthopedic hospitals and schools.
Tuberculosis sanatoria and preventoria.

The extension of occupational therapy within recent years was demonstrated by the program of this meeting. Methods and results were reported by Mr. Asa S. Bacon, Superintendent, and Miss Winifred Brainerd, Chief Occupationalist of the Presbyterian Hospital, Chicago; Miss C. Helen Mowat of the Toronto Curative Workshop; Mr. Eugene C. Foster, Director of the Indianapolis Foundation; Mrs. William H. Marshall from the hospitals of Honolulu (illustrated by moving pictures); Dr. William A. Bryan, Superintendent, and Miss Olive M. Caldwell, of the Worcester (Mass.) State Hospital; Dr. Kenneth J. Tillotson, Superintendent, and Miss Frances Wood, Chief Occupationalist, of McLean Hospital; Dr. George H. Stevenson, Superintendent of the Ontario Hospital, Whitby; Miss Marguerite Emery of Mt. Sinai Hospital, New York; Dr. Bernard T. McGhie, Director of Hospital Services in the Ontario Department of Health; Miss Irene Obrock of the University Hospital, Oklahoma City (illustrated by moving pictures); Miss Jean Hampson of the Wellesley Orthopedic School in Toronto (illustrated); Miss Lillian Spencer of the Orthopedic Hospital of the University of Chicago; Mrs. E. M. McNally of the Junior League of Indianapolis (illustrated by moving pictures); Dr. David A. Stewart, Superintendent of the Manitoba Sanatorium;

Mr. Edward Hochhauser, Director of the Altro Workshops, New York City; Miss A. Edna Buvens, Chief Occupationalist of the U. S. Veterans Hospital at Rutland Heights, Mass.

The session devoted to mental hospitals was probably the outstanding one of the meeting. Dr. Bryan's paper, read in his absence by Miss Constance Garrod, of the Boston School of Occupational Therapy, dealt with the psychological conditions in which occupational treatment is most effective and results most likely to be lasting, paying particular attention to the factor of psychic fatigue.

Miss Caldwell discussed the curative effects of occupational therapy through the socializing of patients and the development of group solidarity in the workshops, which tend to counteract the noxious isolating influence of the psychosis.

Dr. Tillotson stressed the fact that the individualizing method which today is regarded as essential in the treatment of mental patients, should be carried over into occupational therapy, which he considers the most valuable curative means available to the mental hospital.

Miss Wood read a scholarly paper on the psychology of occupational treatment.

Dr. McGhie in describing the program for occupational therapy in the Ontario Hospital Service, paid tribute to Dr. Charles K. Clarke, first Director of the Canadian National Committee for Mental Hygiene, as having been the first to promote occupational therapy as it is known today in Canadian mental hospitals.

The most notable committee report at this meeting was the one on national registration presented by the Chairman, Mr. T. B. Kidner of New York City. The committee has had this work in hand for several years and promises that early in 1932 there will be published a directory of registered occupational therapists in the United States and Canada.

The proceedings of the Toronto meeting and the papers contributed will be published in *Occupational Therapy and Rehabilitation.*

W. R. D.

Book Reviews.

Die Epileptischen Erkrankungen. Ihre Anatomischen und Physiologischen Unterlagen sowie Ihre Chirurgische Behandlung. By Prof. Fedor Krause und Dr. Heinrich Schum. (*I Hälfte. Neue Deutsche Chirurgie, von Ferdinand Enke, Stuttgart, 1931.*)

This is a rather formidable work which attempts to bring together in two volumes all the data available regarding our present state of knowledge of epilepsy. As far as the first volume is concerned, one can say that the attempt is highly successful, and this regardless of the apologetic remarks of the authors for their inability to include all the latest contributions on the subject while their work was in press.

Krause, a well-known neurosurgeon, adds to the compilation of a vast material from all available sources, his own observations and his surgical experience in the treatment of epilepsy covering more than three decades. In this phase of the subject the second volume, dealing with therapy, which is still in press, will be of particular interest.

It is a very difficult problem to analyze a volume of over 500 pages, where each chapter offers a great wealth of material. The historical chapter, gives interesting facts regarding the recognition of epileptic disorders by such ancient peoples as the Incas and Egyptians, the early Romans and other nations of antiquity. It is a pity, however, that in an exhaustive study of this kind, no reference is made to the Jurisprudence of Babylon, which took its inception from the Code of Hamurabi 2080 B. C., where definite reference is made to epilepsy. References to epilepsy are also to be found in the ancient Hebrew writings, dating 2000 years B. C. One finds there such expressions as Nikpheh, meaning the "bent one," referring to the epileptic. If space permitted, one could dilate on this fascinating subject. To these who are interested the writer would recommend an informative article by Prinker, "Epilepsy in the Light of History" (Annals of Medical History, Vol. 1, No. 4).

The chapter on statistical studies contains a survey of data gathered from numerous countries and is illustrated with most comprehensive tables and charts attesting the unusual statistical skill of the authors. It is interesting to note that the admission rate in the epileptic institutions in Germany, as given by the authors, is much higher than the figures given by Binswanger. There are also interesting observations regarding the distribution of convulsions during the various months and hours of the day; and we find, for instance, that the highest peaks are during the months of March and August, and between the hours of 10 p. m. and 6 a. m. No race, no nation seems to remain entirely free from epilepsy, including the savage tribes.

Discussing the pathologic findings in the Ammonshorn as emphasized by Spielmeyer, the authors draw attention to the fact that the hippocampus is particularly vulnerable on account of poor vascularization, and that findings similar to those of epilepsy are seen in this structure in many other organic conditions. The chapter on physiology is one of the most comprehensive, beginning with Kussmaul and passing through the work of all the physiologists, coming down to the modern workers and paying particular attention to the representatives of the American group, *e. g.*, Lennox and Cobb and Pike. The authors do, however, close with Binswanger's assertion, that the cortex is the point of origin for the original discharge of the classical convulsion although it is not always the only site of origin.

In a cursory review justice cannot be done to this monumental work. It is the hope of the writer, at some future date, to make a more detailed analysis, which this work deserves. Meanwhile, it is highly recommended to all students of epilepsy, to whom it offers a copious source of information as well as inspiration for further research.

J. Notkin.

Schizophrenia (Dementia Præcox). An Investigation of the Most Recent Advances. Volume X, Association for Research in Nervous and Mental Diseases, pp. 246. (The Williams & Wilkins Co., Baltimore, 1931.)

The volume contains the proceedings of the Association for Research in Nervous and Mental Disease which met in New York in December, 1929. The second day of the meeting was devoted to the discussion of schizophrenia, the first day having been given to the consideration of epilepsy.

The work represents the progress made in the study of schizophrenia since 1925, when a similar colloquium was held in New York. On the whole one cannot say that the progress has been particularly spectacular but it does show that spasmodic efforts, here and there, have been made in investigation of schizophrenia. It also shows, however, that there is no place in America where real comprehensive continuous effort has been made in the study of this disease. It is quite obvious that the research facilities in psychiatry are ridiculously pitiful when one compares them with the research going on in the fundamental biological sciences or internal medicine. The statement is made that psychiatry lacks a trained scientific personnel to carry on a comprehensive research program. As a matter of fact, this is not exactly true. Excellent men with training and vision, who make a notable beginning in research, are gradually forced into clinical work for the simple reason that the opportunities are very limited and the future uncertain.

The volume opens with a report by Malamud and Rothschild on the study of the barrier between the blood and the cerebrospinal fluid. In some two hundred cases of schizophrenia sixty percent showed a bromide ratio above, 3.20 *i. e.*, there was a definite decrease in the permeability of the barrier between the blood and cerebrospinal fluid. The authors were not able to establish any definite correlation between the various clinical types and the specific resistance to the passage of bromides into the spinal fluid.

Walter Freeman discusses the deficiency of catalytic iron in the brain in schizophrenia. Inasmuch as the deficient oxidation is possibly responsible for the stuporous manifestations in schizophrenia and iron is the chief factor concerned with oxidation, its quantitative determination ought to be very important. For some reason the author is under the impression that he is the first to undertake quantitative chemical studies of the brain since Breed's work in 1851. Palladin in Kharkov and Serjesky of Moscow have made detailed and comprehensive studies, using exactly this method. Freeman finds a definite deficiency of iron in the cortical ganglia cells in schizophrenia and offers the theory that deficiency of the catalytic iron is responsible for some of the mental symptoms.

Trentzsch, starting with the premise advanced by the St. Elizabeth School of Psychiatry that there is a fundamental biological inferiority of the circulatory system in schizophrenia, reports his results with various neurocirculatory tests in pathological states and also in a group of control cases. He believes that by his tests he can demonstrate a weak neurocirculatory system which is more liable to break down under various stresses and strains than a normal one.

Campbell in a paper on the observation of the rôle of environmental factors in schizophrenic conditions points out that the dynamic influence of the environment on personality cannot always be demonstrated by a strictly formal statistical approach. Bowman brings out very interesting data of a purely statistical character on the personality of schizophrenic patients. Both papers are a part of a very comprehensive research which has been going on for several years at the Boston Psychopathic Hospital, subsidized by a special grant, which will eventually yield very important material on methodology of research involving social psychiatric data as well as on the extent to which the statistical methods can be applied to psychiatric material.

Very interesting material is presented by Jelliffe in his paper on vigilance, the motor pattern, and inner meaning in the behavior of some schizophrenics. In the discussion of the paper Dr. White remarks that the psychiatrists are the poets of medicine, which certainly applies to the author.

Spielmeyer presents a remarkably lucid paper on the problem of anatomy of schizophrenia, frankly confessing the lack of specificity in the pathological histology of this disease. Harry Stack Sullivan in discussing the relation of the onset to outcome of schizophrenia offers a very interesting point of view on the psychopathology of the insidious as contrasted with the acute onset. One often gets many more ideas from a free discussion than from a formal presentation, and this holds particularly true of this paper. One cannot help agreeing with Sullivan when he states in the discussion that the onset of schizophrenia can frequently be divided into two stages. "A considerable number of people experience the first step towards schizophrenic phenomena a considerable time before the psychosis makes its appearance. The interval between the initial stadium and the appearance of psychotic experience may be a matter of moments or the matter of a lifetime. I call the first stage the collapse of the individual world synthesis."

In a very comprehensive paper on prognosis in schizophrenia, Strecker presents a series of twenty-five recovered schizophrenics who have been well for periods varying from three to twelve years. This series, well documented and analyzed, presents a body of sound clinical observations which provides a good deal of optimism for anyone dealing with schizophrenics. It is to be regretted that the author has not discussed the therapy or the mechanism of recovery. He makes a very important observation when he states that what reality has to offer to the patient has a great deal to do with the attempt of the patient to get well. There are some situations which are so hopeless that the pathological adjustment is preferable. The prognostic criteria hang largely on the personality of the patient, the factors of toxicity and exhaustion, the precipitating factors and the mode of onset.

Zilboorg discussed the problem of affective re-integration in the schizophrenias. He touches the fundamental issue with the statement that the schizophrenic retains his emotions but they seem to be frozen and completely separated from the rest of his life. The problem of therapy is how to bring these emotions out in response to the ordinary life situations. He believes that psychoanalysis, in virtue of the special method it employs, can actually do it. He illustrates this point with a case which unfortunately does not prove his main thesis, and which, as a matter of fact, throws very little light on the problem.

William J. Bleckwenn reports the results from the use of sodium amytal in catatonia. After the administration of the drug the patients emerged from their stupor for periods lasting two to fourteen hours, after which they lapsed back into their original condition. What exactly takes place when the drug is administered is the problem of biological chemistry.

The volume closes with a very intelligent and timely article by Hinsie criticising the various modes of therapy and the claims of recovery in schizophrenia. He points out that before any therapeutic claims are made one should determine the ratio of spontaneous recoveries which is not usually taken into consideration by the enthusiastic therapists.

The volume is very well edited, the papers are documented with valuable bibliographies, and there is a good index.

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A Psychiatrist's Anthology. By LOUIS J. KARNOSH, M. D. (*Cleveland: The Occupational Therapy Press, 1931.*)

From the apparently endless flux of books on "pop"-psychiatry and commercial psychology, it is a delight to turn to an example of splendid bookmaking, "the product of the avocational moments of a psychiatrist who spends them with his patients in occupational therapy."

As the title suggests, this slender, privately printed, de luxe quarto volume presents rather the artistic than the strictly scientific interpretation of pathological mental states; although the author's word-pictures, and par-

ticularly his remarkable sketches, justly epitomize the clinical conditions selected for the anthology.

In each of the six tableaux—delirium tremens, general paresis, melancholia, schizophrenia, paranoia, senile dementia—subjective and objective are ingeniously combined. In the foreground appears the patient, true to type; while in the background is pictured his state of consciousness. The clinician will recognize the verisimilitude of these representations. The accompanying text contains transcripts of patients' talk, presenting vividly in a few paragraphs in each case the essence of their thinking and feeling.

Not the least interesting feature of this book is that it is almost wholly the work of the author's own hands. The typography and printing, sketching, offset printing and illuminating were done by him, and the volume bears the imprimatur of the Department of Occupational Therapy in the Psychopathic Division of the City Hospital of Cleveland.